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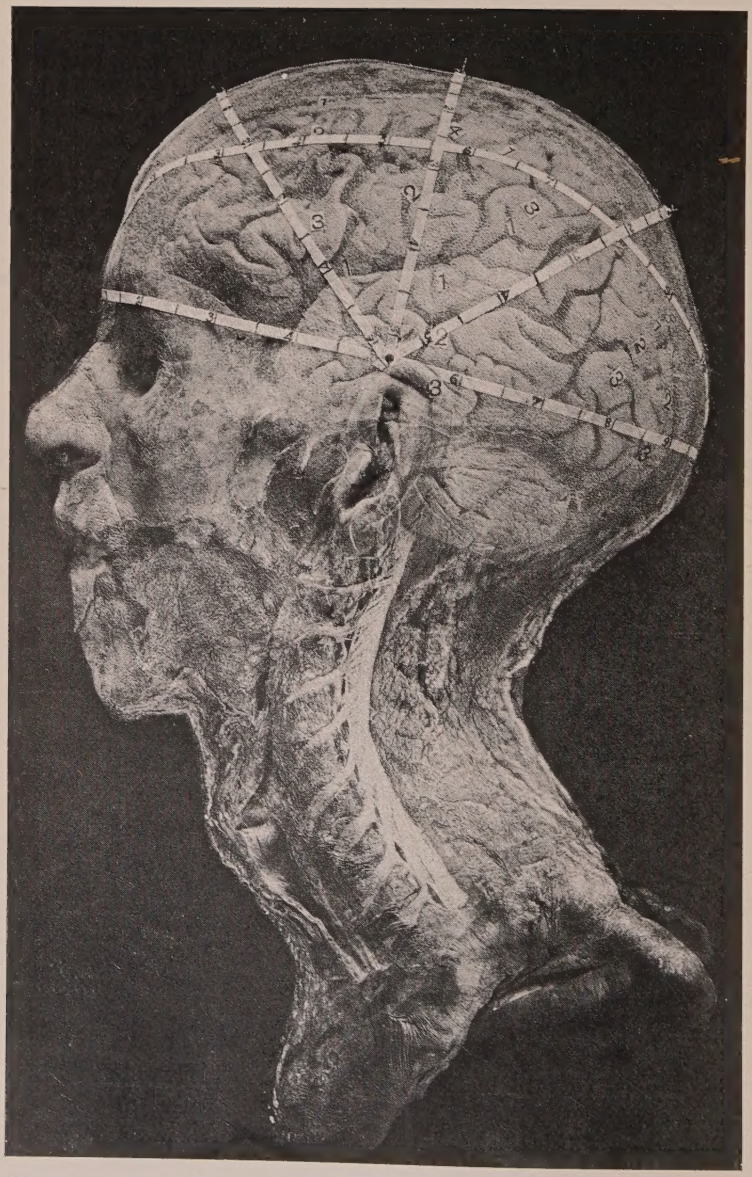

FIG. 210.—COMPOSITE PHOTOGRAPH SHOWING RELATIONS OF CRANIAL SURFACE TO THE FISSURES AND CONVOLUTIONS (ALEC FRASER).

TEXT-BOOK

OF

NERVOUS DISEASES

BEING A COMPENDIUM

FOR THE USE OF

Students and Practitioners of Medicine

BY

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WITH TWO HUNDRED AND TEN ILLUSTRATIONS.

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PREFACE.

It is the object of the author in this treatise to present the science of neurology in a concise yet as far as possible complete form. Each subject has been taken, all the available facts regarding it ascertained, the writer's own experience collated, and with the data thus gathered the chapters have been written. The labor involved in such a task has been very great, but I am encouraged to believe that the result will be a useful one; for the work does not compare or compete with the large treatises which are already in the field nor with the smaller introductory text-books, but I have tried to furnish a book which will be suitable for the student and practitioner and not valueless to the specialist.

The extreme importance of a knowledge of anatomy has led me to pay especial attention to furnishing in a condensed form the most recent accessions to our knowledge of this subject. Starting with the facts that can be gained in ordinary anatomical works, the student can, I believe, acquire a good idea of modern neuro-anatomy with the help of the anatomical chapters given here.

In the classification of nervous diseases and the description of their pathology, I have tried to apply the modern knowledge of general pathology as modified by bacteriology. This I have done conservatively, yet not less than in my opinion is absolutely demanded. A good deal of havoc will be wrought eventually in our conception of the nature of nervous diseases by the newer pathological doctrines; I have made as little change as was consonant with undeniable facts.

The limits placed upon me have made it impossible to furnish a bibliography or to give due credit to every original investiga-

tor. Full references to literature are to be found in the works of Hirt, Erb, Seeligmüller, Ross, and Gowers.

In many topics I have been much helped by valuable monographs of my American colleagues. While a part of these are credited to their proper source in the text, I feel that I ought to refer here to some of the articles that have been of special service to me. They include monographs on Spinal and Brain Tumors by Mills and Lloyd; on Cerebral Palsies of Children by B. Sachs; on Muscular Dystrophies and Writers' Cramp by G. W. Jacoby and by M. Lewis; on Aphasia, Cerebral and Spinal Localizations by M. Allen Starr; on Cranial Temperatures and on Neurasthenia by L. C. Gray; on Degenerative Neuritis by W. H. Leszynsky; on Poliomyelitis by Wharton Sinkler; on Craniometry and Cranial Deformities by F. Peterson and by E. D. Fisher; on Angioneurotic Œdema by Jos. Collins; on Brain Tumors by P. C. Knapp, and on Sclerosis of the Cord by J. J. Putnam. I am indebted to Tourette's recent treatise on hysteria, to that of Fèrè on epilepsy, and to the annual volumes of Bourneville on these subjects. The masterly lectures of Charcot and the treatises of Ross, Gowers, Hammond, Hamilton, and Putzel have necessarily been freely used. In the anatomical part I have used the works of Edinger, to whose courtesy I am particularly indebted, the treatise of Obersteiner, and many monographs by Golgi, Marchi, Cajal, His, Waldeyer, and others. My own work in teaching anatomy and pathology has enabled me to do more than present a compilation.

I must finally express my thanks to my publisher, Mr. W. H. S. Wood, for his patience and helpful generosity in my efforts to make my work a production that would be creditable to American neurology.

To the Student.

As a special text-book the present work will be used by two classes of readers, one consisting of those who simply consult it for reference in connection with their cases, the other composed of students who desire to ground themselves systematically in a knowledge of neurology. To this latter class I venture some advice as to the method they should pursue. Neurology is a

difficult branch of medicine to master, nor is there any royal road to it. Still, it can be made comparatively easy if its study is undertaken in a proper and systematic way.

In using the present work, the student should first refresh his general knowledge of nervous anatomy as furnished in ordinary text-books. He should then go carefully over the anatomical descriptions here given of the general structure of the nervous system and of that of the nerves, spinal cord, and brain. A thorough knowledge of anatomy and physiology makes clinical neurology comparatively easy, and in fact reduces much of it simply to a matter of logical deduction.

The student should next master the general facts of nervous pathology, symptomatology, and etiology, for he will find common laws underlying apparently the most varying phenomena. Finally, he must begin to study the special diseases. The number of these is very great; in the present work I have described 176. Many of these are rare, and it would be wrong for the student to burden his memory with the details about them. He need know only of their existence and general physiognomy. There are, however, according to my enumeration, about 65 nervous diseases which are either very common or extremely important, and it is these that the student should master and make part of his working knowledge. Since the distribution and names of the common and rare diseases may be a useful guide, I append here a table and a list:

	Peripheral.	Spinal Cord.	Brain.	Functional.	Totals.
Common and important nervous diseases.....	31	13	12	10	65
Rare.....	56	27	16	11	111
	87	40	28	21	176

The common or important peripheral nervous diseases are:

General.—Neuritis, multiple neuritis, degeneration, neuralgia, paræsthesia (5).

Cranial.—Anosmia, optic neuritis, optic atrophy, ptosis, ophthalmoplegia, abducens palsy, headache, migraine, trigeminal neuralgia, facial spasm, facial palsy, tinnitus, vertigo, ageusia, wry-neck (16).

Spinal Nerves.—Cervical neuralgia, hiccough, brachial palsies, single and combined, brachial neuralgia, intercostal neuralgia, herpes zoster, lumbar neuralgia, sciatica, leg palsies (10).

Spinal Cord.—Spina bifida, hemorrhage, pachymeningitis, leptomeningitis, poliomyelitis, transverse myelitis, acute and chronic, secondary degenerations, locomotor ataxia, the progressive muscular atrophies, bulbar palsy, muscular dystrophies, spinal irritation (13).

Brain.—Malformations, hyperæmia, pachymeningitis, leptomeningitis, simple, tubercular, and epidemic, abscess, hemorrhage, embolism, thrombosis, children's palsies, syphilis (12).

Functional.—Epilepsy, hysteria, the tics, chorea, tetanus, neurasthenia, spermatorrhœa, exophthalmic goitre, occupation neuroses, paralysis agitans (10).

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Diseases of the Nervous System.

CHAPTER I.

GENERAL ANATOMY OF THE NERVOUS SYSTEM.

I SHALL describe here, first, the fundamental constituents of the nervous system, and next the general facts with regard to their arrangements.

The nervous system is derived from the epiblastic layer of the developing ovum, and its constituents are modifications of epithelial cells. These cells in the embryo are of two kinds: neuroblasts, which develop into nerve cells and fibres; and spongioblasts, which develop into a supporting structure called neuroglia (His).

The nervous system is composed of—

(*a*) Nervous tissue proper, viz.: nerve cells and nerve fibres, forming together nerve units or *neurons;* and neuroglia.

(*b*) Non-nervous tissue, viz.. connective tissue, blood-vessels, lymphatics, and epithelium.

These tissues are united together to form a *central nervous* system, consisting of the brain and spinal cord, and a *peripheral nervous* system, consisting of nerve fibres, collections of nerve cells called nerve *ganglia*, and structures attached to the terminations of the nerves, called end-organs.

THE NERVE CELLS OR GANGLION CELLS. — *Shape.* — They are of various shapes, differing in different parts of the nervous system. They are oftenest irregularly spheroidal, pyramidal, spindle or flask shaped. They give off processes or poles, and are classified usually in accordance with the number or arrangement of these. There are four kinds, viz.: (1) multipolar; (2) bipolar; (3) unipolar; and (4) small nuclear. The multipolar cells are the common type.

Size.—They vary much in size. The larger cells are about $\frac{1}{250}$ inch, the smallest $\frac{1}{3500}$ inch, in diameter.

Structure.—The nerve cells are composed of a protoplasmic body, containing a nucleus and nucleolus. The protoplasm of the body appears granular, and sometimes striated or reticulated.

It often contains yellowish pigment. The nucleus is apparently reticulated. The processes are given off in various directions. In all developed nerve cells, one or more of the processes is prolonged; it then receives a myelin sheath and becomes a nerve fibre. This is called the *nervous* or *axis-cylinder process.* All *nerve fibres are simply prolongations of nerve-cell processes* and are really parts of nerve cells. The nervous process sends off

FIG. 1.—MULTIPOLAR CELL OF FIRST OR MOTOR TYPE (MARCHI), showing nervous process and collaterals.

lateral fibres at right angles as it passes out, and farther on, when it becomes a nerve fibre, it sends off other processes; these are called *collaterals* (Golgi, Cajal). It ends finally in a brush-like fibrillation called the "end-brush." The other processes of nerve cells are called *protoplasmic.* They connect with the blood-vessels and lymphatic spaces and, perhaps, have a nutrient function. They do not, at any rate, connect with other nerve-cell processes. Multipolar cells are said (Golgi, Von Monakow) to be of two types—motor and sensory. In the cells of the motor

type the nervous process passes directly into the axis cylinder of a nerve. In the sensory type the process passes first into a plexus of fibres, out of which a single nerve finally emerges.

Some multipolar and most bipolar cells give off two nervous processes; one of these may be medullated, the other not. Many nerve cells of the peripheral ganglia have a membranous sheath or capsule continuous with the sheath of the nerve. The cells

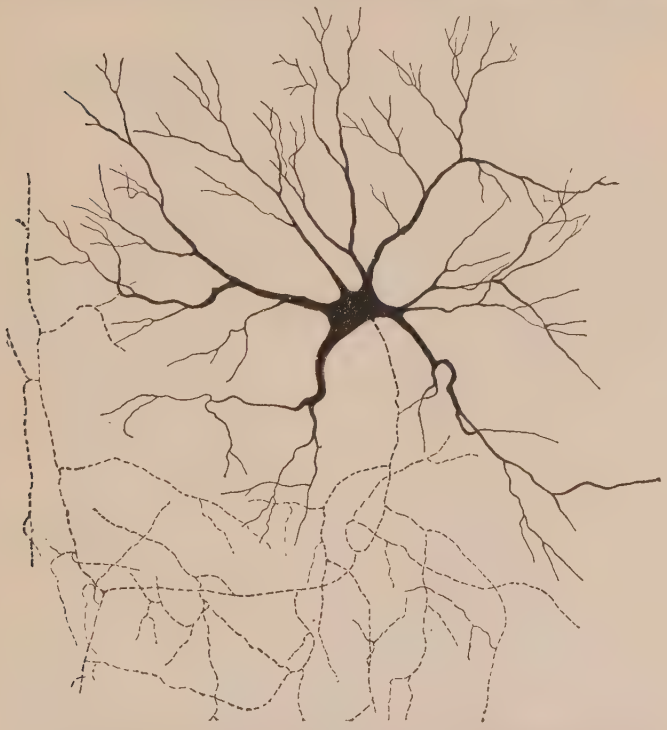


FIG 2.—MULTIPOLAR CELL OF SECOND OR SENSORY TYPE (MARCHI), showing nervous process and collaterals.

of the central nervous system have no capsules. The shape and structure of the various cells are best shown by the accompanying cuts. The location of the different kinds of cells is as follows:

Multipolar cells are found in the cortex cerebri, basal ganglia, and central gray of the spinal cord and medulla, and in some peripheral ganglia.

Bipolar cells in Clark's column and intermediate gray of the cord, and in the posterior spinal ganglia.

Unipolar cells in the posterior spinal and sympathetic ganglia.

Small nuclear cells and flask-shaped or *Purkinje's cells* are found in the cerebellum.

Besides these, there are described, in the brain cortex, angular, granular, pyramidal, globose, and spindle cells.

THE NERVE FIBRES of the nerve centres are found chiefly in the white tissue or white matter. In the periphery they form the nerve proper of gross anatomy.

The peripheral nerve is composed of bundles of nerve fibres

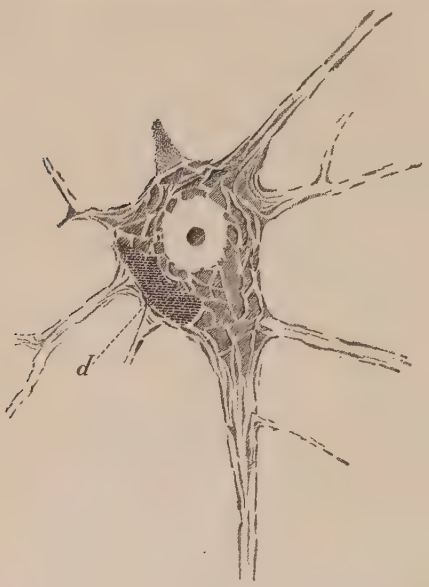


FIG. 3.

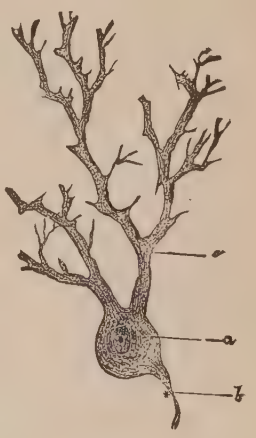


FIG. 4.

FIG. 3.—MULTIPOLAR CELL, showing processes, reticulum, nucleus and nucleolus. *d*, Pigment (Nissl).

FIG. 4.—MULTIPOLAR (PURKINJE) CELL FROM CEREBELLUM. *a*, Body; *b*, nervous process; *c*, protoplasmic processes.

called nerve fasciculi. It is surrounded by a connective-tissue sheath called the sheath of Henle, or *epineurium*. From this sheath, connective-tissue fibres pass in and surround the fasciculi. The sheath of the fasciculus is called the *perineurium*. From the perineurium, strands of connective tissue run in among the ultimate nerve fibres, forming the *endoneurium*. Lymphatic spaces lined with endothelium exist in the layers of the peri- and endo-neural sheaths. In the nerve centres, the nerve fibres have no regular sheaths, but are supported by a connective-tissue and neuroglia framework. The *nerve fibre* is a long fine strand of

tissue varying in diameter. It may be white or gray according to its structure. It is composed from within out of (1) an axis cylinder, (2) a myelin sheath, and (3) a neurilemma. (1) The *axis cylinder* is the essential part of the nerve. It is the prolongation of one of the processes of a nerve cell and consists of protoplasm. It is itself made up of fine fibrillæ (primitive fibrillæ)

FIG. 5.—UNIPOLAR CELL. *a*, Body; *b*, capsule; *c*, process.

which run longitudinally. By means of reagents, a transverse striation can be seen also. (2) The *myelin sheath*, *medullary sheath*, or sheath of Schwann surrounds the axis cylinder. It is composed of a semi-fluid, fatty substance, which chemically consists of lecithin, neurin, and some cholesterin. It varies much in thickness, and this is the principal cause of the different sizes of

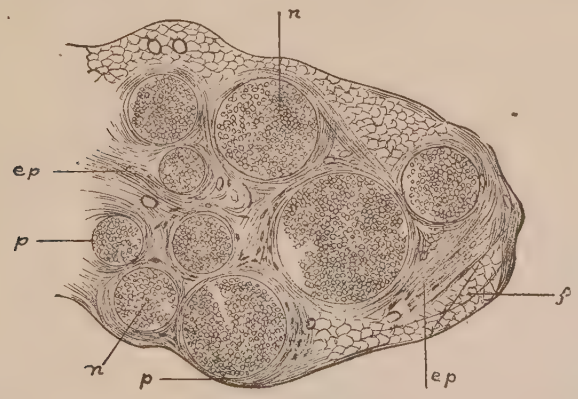


FIG. 6.—FROM A TRANSVERSE SECTION THROUGH THE SCIATIC NERVE. *ep*, Epineurium; *p*, perineurium; *n*, nerve fibres constituting a nerve bundle or fasciculus in cross-section; *f*, fat tissue surrounding the nerve (Klein).

nerves. The myelin sheath is interrupted at regular intervals by constrictions called the "nodes of Ranvier." These constrictions involve the myelin sheath alone. The axis cylinder passes through and the outer sheath (neurilemma) passes over it. There is a little granular matter at the point, called intercellular cement. The nerve fibres, if they divide, always do so at a node (Fig. 10). The

part between two nodes is called a nerve segment. In each segment there is an oval nucleus imbedded in the myelin sheath. The nodes are about 1 mm. apart.

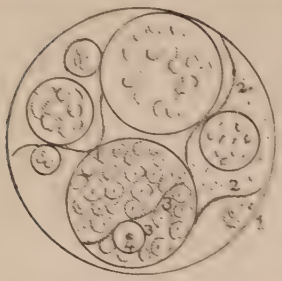


FIG. 7.—DIAGRAM SHOWING THE DIFFERENT PARTS OF THE NERVE. From without inward we have: 1, Nerve with epineurium; 2, nerve fasciculus, with perineurium; 3, nerve fibre and endoneurium; 4, neurilemma, myelin sheath, axis cylinder, primitive fibrillæ.

The myelin sheath is probably developed, like the axis cylinder, from the epiblast, and is closely related nutritionally to the axis cylinder, which it protects and isolates. Between it and the axis cylinder Mauthner describes a membranous sheath (**axis-cylinder sheath**). Another sheath is said to be between it and the neurilemma (medullary sheath). By means of certain reagents, oblique lines (incisures of Schmidt) or a reticular appearance may be developed (network of Gedvelst). These appearances are, perhaps, artificial. (3) The *neurilemma* or *primitive sheath* is a delicate homogeneous covering forming the outermost sheath of the nerve. It is of connective-tissue origin. The sheath is absent in the fibres of the central nervous system and in some fibres of the periphery.

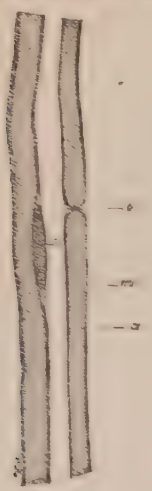


FIG. 8.

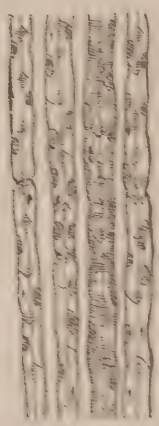


FIG. 9.

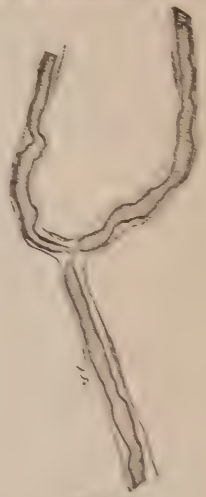


FIG. 10.

FIG. 8.—MEDULLATED NERVE FIBRE. *a*, Axis cylinder; *b*, nucleus; *c*, medullary sheath; *c*, node of Ranvier.

FIG. 9.—MEDULLATED NERVE FIBRE, showing axis cylinder, nuclei of medullary sheath, nodes, and oblique incisures of Schmidt.

FIG. 10.—MEDULLATED NERVE FIBRE, showing mode of division.

Variations in the Types of Fibres.—In accordance with the arrangement of the sheaths of the nerve fibres, several kinds are described. The principal types are the *medullated* and *non-medullated.*

Medullated nerve fibres make up the bulk of the white matter of the brain and cord and cerebro-spinal nerves. They consist of a myelin sheath and axis cylinder, and may or may not have a neurilemma.

Fibres with myelin sheath, but without a neurilemma, make up the white matter of the central nervous system.

Non-medullated fibres, or fibres of Remak, occur principally in the sympathetic system, but they are also found in the cerebro-spinal nerves. They are grayish and faintly striated, and consist of axis cylinders, with a thin, homogeneous, nucleated sheath lying directly upon them. This sheath, however, cannot often be demonstrated (Schaefer).

Naked axis cylinders are found in the peripheral terminations of nerves as well as in the brain and cord.

Size.—The nerve fibres are of two kinds as regards size. The small fibres are about $\frac{1}{12000}$ inch in diameter, the large $\frac{1}{1200}$ inch. The small fibres are connected with smaller cells, and either run a shorter course or are distributed to the involuntary muscular fibres of the blood-vessels and viscera.

The nerve fibres of the central nervous system give off "collaterals," *i.e.*, small medullated fibres branch out at right angles and pass off to make connections in an unknown way with nerve cells or other fibres.

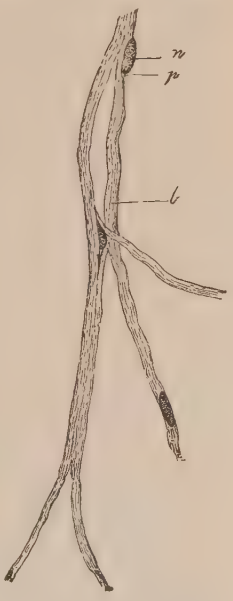


FIG. 11.—NON-MEDULLATED NERVE FIBRE. *n*, Nucleus; *b*, striations.

These fibres and those of the nerve roots have no neurilemma, but are surrounded with neuroglia tissue. They always end in a brush-like plexus of primitive fibrils, which surrounds the nerve cells in a network.

The peripheral nerve fibres, except the optic, have no neuroglia; they terminate in fine fibrillæ among epithelial cells, or in special end organs.

Connections of Nerve Cells and Nerve Fibres.—One nerve cell is never connected directly with another, so far as anatomical investigation can show. One nerve process becomes an axis cylinder, receives a myelin sheath, gives off collaterals, and finally

breaks up into a fibrillary "end-brush" surrounding a cell, but not passing into it. There is physiological, but no apparent anatomical continuity (Fig. 12).

The cell and its nerve fibre and end-brush form together the nervous unit or neuron. The typical arrangement of a neuron is shown in Fig. 12.

FIG. 12.—DIAGRAM SHOWING THE NEURON AND MODE OF CONNECTION BETWEEN NERVE UNITS THROUGHOUT THE NERVOUS SYSTEM. 1, Nerve cell; 2, nervous process; 3, collateral; 4, end-brush; 5, nerve cell.

The Neuroglia. — The supporting tissue of the peripheral nerves is connective tissue only; that of the central nervous system is connective tissue and, in addition, a peculiar substance called neuroglia. The neuroglia or supporting tissue of the nervous centres is derived from the epiblast. It is composed of cells with very numerous and finely ramified processes, which make a supporting network about the nerve cells and fibres. The neuroglia cells are known as "spider cells" or Deiter's cells. They differ somewhat in size and shape, but not in general characteristics. The fibrillary processes form a felt-like network; and in regions where there is much neuroglia tissue, this looks like a homogeneous matrix. It is, however, made up of the fine fibrils. These connect with the walls of the blood-vessels. In inflammatory conditions the cells multiply, swell up, and assist in carrying off irritating products (scavenger cells of Lewis). Neuroglia tissue is richly deposited about the central canal of the spinal cord, in the substantia gelatinosa, beneath the ependyma of the ventricles and beneath the pia mater of the brain and cord. The epithelial cells of the central canal and ependyma of the ventricles send down fine processes which form a minor part of the supporting framework.

THE NON-NERVOUS TISSUES. — *The Blood-Vessels.* — The peripheral nerves are richly supplied with blood. Each nerve re-

ceives arterial supply from many different branches, but always from the same general source. The artery passes to the nerve sheath obliquely, then divides dichotomously and sends branches a long distance up and down on the sheath. It may pierce the sheath, however, first, and then divide, as above described. The dichotomous branches send off arterioles and capil-

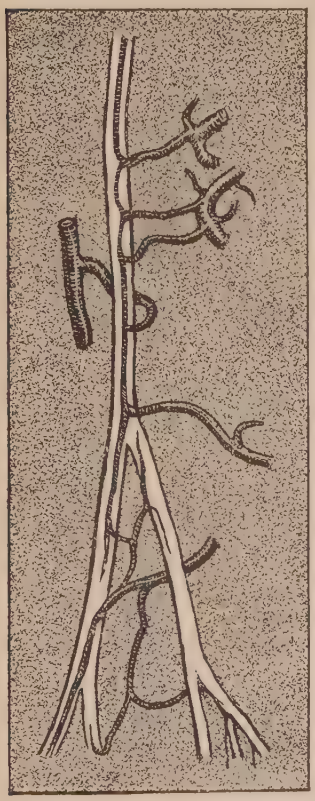


Fig. 13.

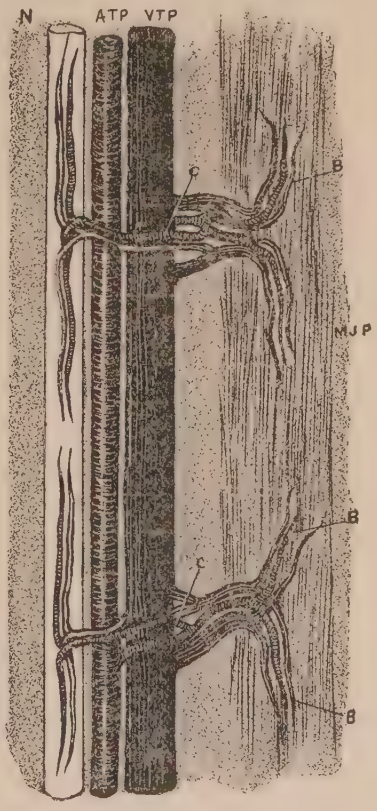


Fig. 14.

Fig. 13.—Arteries of Sciatic Nerve (Quénu and Lejars).

Fig. 14.—Venous Supply of Posterior Tibial Nerve. *N*, Nerve; *ATP*, artery; *VTP*, vein; *MJP*, muscle; *B*, veins to muscle; *C*, *B*, veins to nerve (Quénu and Lejars).

laries, which form plexuses about the nerve fascicles. These are "the interfascicular arcades." The arteries subdivide in such a way as to prevent sudden impact of a large blood stream into the tissue of the nerve. In this respect the nerve circulation resem-

bles that of the brain and cord. The veins subdivide dichotomously, like the arteries. They freely anastomose with the muscular veins, so that muscular action helps nerve circulation. The veins of the superficial nerves connect with those of the deep nerves.

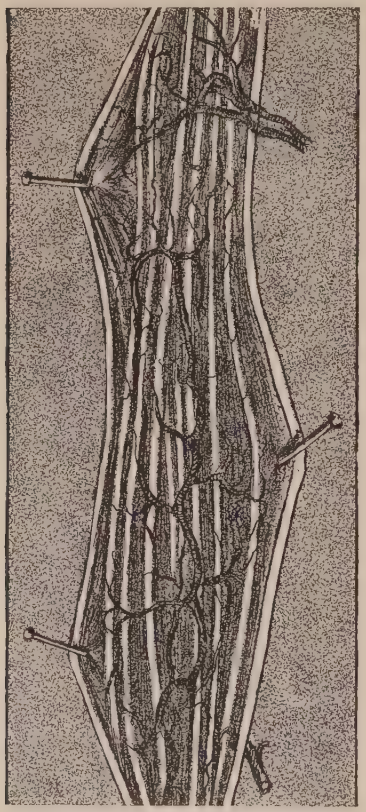


Fig 15.—Interfascicular Distribution of Arteries (Quénu and Lejars).

The blood-vessels of the spinal cord and brain will be described later.

Lymphatic vessels and spaces are found in the epineurium and perineurium. There are no distinct lymphatics in the fasciculi, but lymph spaces probably exist.

The General Arrangement and Functions of the Nervous System.

Having described the component parts, I shall now show the way in which these parts are arranged to form the nervous system.

The nervous system, as already stated, consists of—

I. A central part, composed of the brain and spinal cord.

II. A peripheral part, composed of nerves, peripheral ganglia, and end organs.

I. The nerve centres are made up of gray and white matter, inclosed in membranes called the meninges. The gray matter consists chiefly of nerve cells arranged in groups or masses. The white matter consists of nerve fibres arranged in tracts, strands, or columns.

The gray matter is deposited in five principal places.

The myelon, or central gray matter of the spinal cord, and brain axis, extending to the tuber cinereum.

The corpora dentata, olivary bodies, and red nuclei.

The great basal ganglia, viz., corpora striata composed of lenticular and caudate nuclei, the optic thalami, and the corpora quadrigemina.

Cortex of cerebellum.

Cortex of cerebrum.

Besides these collections of gray matter, there are minor ganglia in the pons, cerebellum, and cerebrum, which will be referred to later. The white matter connects these various ganglionic deposits of gray matter with each other and the periphery.

II. The peripheral nervous system is usually described as consisting of a cerebro-spinal and a sympathetic part. Both portions, however, are alike made up of nerve fibres derived from and dependent on the brain and cord. Both are composed of nerve fibres connected in their course with peripheral ganglia and terminating in end organs. These peripheral ganglia consist of three sets: (1) The spinal ganglia, on the posterior roots; (2) the vertebral or sympathetic ganglia, connected chiefly with the anterior roots; and (3) the peripheral ganglia proper, including vasomotor and visceral ganglia, with which fibres from both roots are connected.

The nerves which run between centre and end organs carry nerve impulses both ways. They are, therefore, divided into the *afferent*, centripetal or in-going, and *efferent*, centrifugal or out-going. The old division into motor and sensory nerves will not answer, for we know that there are many out-going nerves which are not motor. The *afferent* nerves are—

1. The sensory, including:

Nerves of general sensation.	Pain nerves or pathic nerves, heat and cold or thermic nerves.
Nerves of special sensation.	Tactile, including: Contact, Pressure, Locality.
	Nerves of muscular sense.
	Nerves of special sense of smell, sight, taste, hearing, and space.

2. Excito-reflex nerves.

The *efferent* nerves are—

1. Motor nerves, going to voluntary or striped muscles, heart muscle, smooth muscle, including the vaso-constrictor and dilator nerves.

2. The secretory. These act upon glands. Impulses to the blood-vessels (vasomotor) generally accompany the secretory impulses.

3. The trophic.

4. The inhibitory. These nerves control muscular movements, secretion, perhaps also nutrition.

We must admit that afferent and efferent impulses take place also between end organs and certain (so-called) sympathetic ganglia. In other words, the cerebro-spinal axis is not always the

only centre. But these subordinate and peripheral centres are normally in connection with the spinal cord and may be influenced by it or by higher parts.

There are also

Intercentral or commissural nerves, which connect different parts of the cerebro-spinal system together. Some of these connect symmetrical parts on each side together. They have co-ordinating function. Others connect higher with lower centres. These latter are made up of ascending and descending fibres. Higher centres send down impulses by the latter, which may stimulate or inhibit lower centres. In the peripheral nervous system we have also *end organs*. These are delicate and in some cases complex arrangements of the nervous and other tissue at the periphery of the nerves. Their object is to allow the nerves to be irritated by special *stimuli* which would not otherwise affect them, *e.g.*, light or sound. Their object is also the proper utilization of efferent impulses upon other tissues.

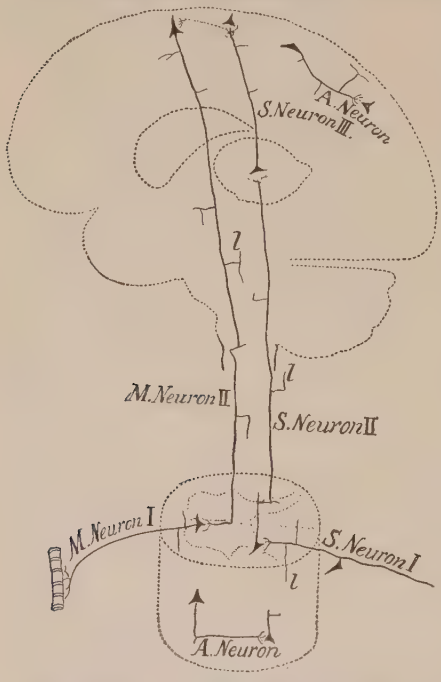


FIG. 16.—DIAGRAM SHOWING THE ARRANGEMENT OF THE NEURONS OR NERVE UNITS IN THE ARCHITECTURE OF THE NERVOUS SYSTEM. *M. Neurons I.* and *II.*, Motor neurons; *S. Neurons I., II., III.*, sensory neurons; *A. Neuron*, associative or commissural neuron.

There are end organs, therefore, for both sensory or afferent and for efferent nerves.

The end organs of the afferent nerves are—I. Eye. II. Ear. III. Taste-buds. IV. Corpuscles in the Schneiderian membrane. V. Various tactile cells and bodies. VI. The space-sense organ.

For the efferent nerves—I. Neuro-muscular corpuscles in the voluntary muscles. II. Local ganglia about the arteries. III. Local ganglia in the glands. Trophic end organs are not known. In many cases the end organ is nothing but the ter-

minal fibres of the nerve. These lose both medullary sheath and neurilemma, leaving only the axillary cylinder. These cylinders split up into terminal plexuses, or else without splitting pass between and around the cells which they are to affect.

The nerves, centres, and end organs thus described may be arranged in mechanisms, each mechanism subserving a special function. These form the *mechanisms of the nervous system proper*, and those of the other organs of the body.

They are as follows:

Cerebro-spinal.
- I. The psychical mechanism or mind.
- II. The automatic mechanisms or the mechanism of inherited and acquired aptitudes.
- III. The sensory mechanism.
- IV. The voluntary motor mechanism.
- V. The reflex mechanism.

Visceral mechanisms.
- The secretory mechanism.
- The trophic mechanism.
- The thermic mechanism.
- The vasomotor mechanism.

Various of the simpler mechanisms are combined to form those more complex. Thus the automatic and psychical mechanisms embrace in their activity other mechanisms of lower grade.

In the same way mechanisms are combined for the regulation of visceral functions. Thus we have—

Compound neuro-mechanisms.
- I. The cardiac neuro-mechanism—a combination of automatic, reflex, inhibitory, motor, and sensory fibres and cells.
- II. The respiratory mechanism.
- III. The mechanisms of the digestive tract.
- IV. The renal mechanism.
- V. The thermal mechanism.
- VI. The skin and sweat mechanism.
- VII. The vesical mechanism.
- VIII. The sexual mechanism.

In the working of these mechanisms the nerve cell is the agent which generates the energy or nerve force, by which impulses are started, controlled, and distributed. The larger the nucleus of the cell in proportion to its protoplasmic body, the more stable or less sensitive the cell. The larger the amount of protoplasm relatively to the nucleus, the more active the discharging power of the cell. The nerve fibres conduct impulses generated by nerves. These impulses travel at the rate of about 100 to 120 feet per second. It is less in visceral nerves (25 to 30 feet per second). There are no electrical currents in normal living nerves (Landois) except when

an impulse travels along them. Then an electrical current travels along with the impulse. It is called the current of negative variation. The irritability or excitability of a nerve is the power it has of responding to a stimulus. When a constant electrical current is passed along a nerve its irritability is modified. This modified condition is called *electrotonus*.

When a nerve impulse passes up an afferent nerve and is then reflected along an afferent nerve, it is called a *reflex action*. The time required for this process is called the *reaction time*. This averages from 0.125 to 0.2 of a second.

CHEMISTRY.—The nervous tissues are alkaline in reaction, but acidity rapidly increases on activity. Acidity is due to lactic acid. It is greater in the gray matter. The percentage of water is shown in the accompanying table, compiled from Halliburton's figures:

Portion of Nervous System.	Percentage of Water.			
	Fœtus.	Age. 20–30.	Age 70–94.	
Gray matter	90	83	84	..
White matter		69	72	74
Nerves	..	..	..	68

Nerves have the least water, gray matter of the brain most.

Inorganic salts vary in amount, but average 0.5 per cent. PO_4 is the largest single constituent of the brain; potassium is next (Geoghegan).

The nervous system is composed of—

1. Proteids, which make up about half the solids in gray matter, one-fourth those in white matter, and one-third those in nerves.

2. Albuminoids, which are neuro-keratin and nuclein.

3. Phosphorized constituents known as protogon and lecithin.

4. Nitrogenous bodies of unknown composition called, 5, cerebrins; 6, cholesterin, which is a monatomic alcohol abundant in white matter; 7, extractives; and 8, inorganic salts.

The following table shows the proportions of the different organic constituents of the brain (Petrowsky):

	Proteids.	Lecithin.	Cerebrin.	Cholesterin and Fat.	Other Organic Matter.	Salts.
Gray matter	55.37	17.24	0.53	18.68	6.71	..
White matter	24.72	9.90	9.55	51.91	3.34	..

CHAPTER II.

GENERAL DESCRIPTION OF DISEASES OF THE NERVOUS SYSTEM.

The Method of Studying Nervous Diseases.

In studying the vital phenomena of the human body, we learn first its normal structure and functions. Then when diseased we note the new phenomena which have developed, the causes which produced them, and the anatomical changes lying back of them; we group our facts and give the disease a name. Finally, we apply the methods by which the disorder can be relieved and future attacks prevented. In fine, we investigate our subject just as we study any branch of natural history. Our study divides itself, therefore, into—

Normal Anatomy and Physiology.

Symptomatology, a study of the morbid phenomena.

Etiology, a study of the causes.

Pathology, under which we include a study of the morbid anatomy and physiology.

Diagnosis, or the method of differentiating the different groups of diseases.

Prognosis, a forecast of the future course of the malady.

Treatment and Prophylaxis.

Again, although nervous diseases exhibit many phases and have many different morbid changes behind them, there are certain features common to all. It simplifies their study, therefore, to learn first what these general features are, just as a person can better survey and plot out a country in detail if he knows certain general facts about its boundaries and topography. Hence I shall first present in a general way an outline of the fundamental facts that touch more or less on all forms of nervous disease. My first chapters will be devoted to—

General Anatomy and Physiology, Symptomatology, Etiology, Pathology, Diagnosis, Prognosis, and Treatment.

The subject of anatomy and physiology has already been briefly presented. We shall now take up

Symptomatology.

I shall begin with a definition of certain terms that are frequently used.

Neurology is the science of nervous diseases.

Psychiatry is the science of mental diseases.

A *neurosis* is a morbid nervous state. The term as sometimes used is restricted to morbid nervous states of functional character. This is not strictly correct, and in speaking of neuroses I give it the general meaning.

A *psychosis* is a morbid mental state, and is nearly identical with an insanity, but has a broader meaning.

The vital activities of the nervous system are shown in the phenomena of the mind, in muscular movements, sensation, control of blood-vessels and secreting glands, in modifications of nutrition, of temperature, and visceral action. The disorder of any one of these functions produces a train of symptoms which receives a name in accordance with its character and class.

Disorders of motion are called *kinesio-neuroses;* those of sensation, *æsthesio-neuroses.*

Correspondingly we have *tropho-neuroses*, *thermo-neuroses*, *vasomotor*, *secretory*, and *visceral neuroses.*

When mental or nervous functions are disordered, there is an increase, decrease, or perversion of the special function or activity. Certain Greek prefixes are used to indicate this. They are: "hyper," which mean excess; "hypo," meaning diminution; "a" or "an," which means absence; and "para," which means perversion.

So we have, for example, hyperæsthesia or excessive sensibility; anæsthesia, which means loss of sensation; paræsthesia, which means perversion of sensation.

From the foregoing it will be seen that the classes or kinds of nervous symptoms are—

1. Mental and cerebral, forming psychoses.
2. Motor and reflex, forming kinesio-neuroses.
3. Sensory, forming æsthesio-neuroses.
4. Trophic, forming tropho-neuroses.
5. Vasomotor and thermic, forming angio-neuroses and thermo-neuroses.
6. Secretory, forming secretory neuroses.

Combinations of these groups of symptoms may affect various organs. They are called mixed neuroses. A disorder may affect certain parts of the body, like the extremities—it is then called an acro-neurosis; or certain functions like the sexual organs—it is then called a sexual neurosis.

Combinations of mental and nervous symptoms form psychoneuroses.

A common and practical division of nervous symptoms is into the objective and subjective.

Objective symptoms are those which can be observed and noted by the physician.

Subjective symptoms are those which are felt by the patient and give no outward sign. Headache is a subjective symptom; atrophy is an objective one.

The particular symptoms which nervous diseases cause are best described and recorded under the several heads given above.

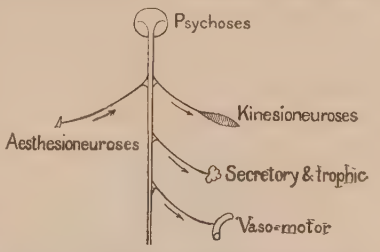


FIG. 17.—DIAGRAM ILLUSTRATING THE PRINCIPLE OF THE CLASSIFICATION OF NERVOUS SYMPTOMS.

1. The *mental symptoms* include all those found in insanity, idiocy, and imbecility, and will not be given in detail here.

The common symptoms met with by the neurologist are mental irritability, depression, emotional excitement, morbid fears, volitional weakness and lack of self-control, persistent or fixed ideas, weakness of memory and of power of concentration, and a tendency to hypnotic and somnambulistic states.

Certain symptoms due to disturbance of brain function are often called *cerebral*, as distinguished from mental. They are: vertigo, disorders of equilibrium, insomnia, somnolence, stupor, coma.

Headache, head pressure, and similar feelings are also often described under the head of cerebral symptoms.

2. *Motor Symptoms.*—The symptoms of disordered motility may be classified as follows:

A. Symptoms of exaggerated motility or hyperkineses.

Tremor: *a*, fibrillary;
b, tremor proper: intentional, or tremor on motion; tremor in rest; constant.

Convulsions or spasms: tonic; clonic; co-ordinate or purposeful.

Choreic movements, convulsive tic, athetosis.

Muscular tension and rigidity, contracture, myotonia, myoclonus.

Forced movements, associated movements.

Exaggerated reflexes, clonus, trepidation.

B. Symptoms of lessened motility.

Paralysis and paresis: { monoplegia, hemiplegia, paraplegia, diplegia.

Loss of reflexes, superficial and deep.

Tremor is the result of a disorder in the tonic innervation of muscles. Muscles are kept normally in a state of slight tension by rhythmical impulses from the brain cortex passing down at the rate of about twelve per second. When the rhythm and force of these normal impulses are interfered with we have tremor.

The simplest form of tremor is one in which the normal tonic impulses have an apparently exaggerated force. This causes a fine tremor of ten to fourteen vibrations per second. When there is an interruption to some of the impulses we have a coarse tremor. Here the vibrations are five to eight per second. It is caused by a partial or complete dropping out of the alternate impulse.

Various technical names are used in describing the tremors.

We have the fine and coarse, as described.

Intention tremor is one that occurs on voluntary movement, and is opposite in kind to the tremor of rest, which decreases or ceases on voluntary effort.

The term paralytic tremor is used to express the tremor of exhaustion, and the term spastic tremor that of exaggerated motility, as in the tremor of a chill. Ataxic tremor is one in which inco-ordinate movements are combined with the tremor.

Fibrillary tremor is a fine twitching of the individual strands or parts of muscles, and occurs usually when they are wasting from lack of neuro-trophic influence.

Convulsions consist of abnormal and exaggerated muscular contractions occurring in rapid succession. Convulsions may be *clonic*, *i.e.*, the muscles rapidly and alternately contract and relax in an exaggerated and irregular way; or they may be *tonic*, *i.e.*, contracted steadily and continuously. When a tonic muscular contraction is painful it is called a *cramp*. Convulsions may be co-ordinate. In this case the patient moves the limbs and body in a more or less purposeful way. He throws himself about the bed, jumps, kicks, strikes, tears the clothes, etc.

Convulsions are usually accompanied with loss of consciousness.

Choreic movements are sudden jerking, twitching movements of different groups of muscles. The movements are purposeless and are not under control of the will.

Convulsive Tic.—When a choreic movement is confined to certain groups of muscles which work together for a common purpose, like those of the face, or eyes, or larynx, it is called a "tic." The movements in the "tics" are more definite in character and

are limited to muscles physiologically grouped for a definite function. Thus we have tics of the muscles of expression, or of respiration, or speech, or locomotion.

Athetosis.—This is a name given by Hammond to a peculiar form of movement characterized by slow, successive flexion, extension, pronation, and supination of the fingers and hand and arm, or of analogous movement of the toes and feet. The motion rarely ceases in waking hours except for a short time. The contractions are forcible, steady, and even, and sometimes painful. The hand assumes characteristic positions.

A *contracture* is a tonic muscular spasm of long duration, *i.e.*, days or months. A contracture may be functional or organic; and in order to test this, one must find whether it ceases during sleep or under an anæsthetic; if so, it is functional (see Hysteria).

Fig. 18.—The Hand in Athetosis (Strumpell).

In forced movements the patient suddenly and involuntarily is thrown forward, sideways, or whirled about in various ways.

Associated movements are those which occur involuntarily in a limb or muscle at rest when the corresponding limb or muscle is moved on the opposite side.

Thus in hemiplegia the movement of the normal arm may excite a movement in the one paralyzed. The patient is given a piece of chalk in each hand, and each hand is placed upon a blackboard lying on the table; attempts at drawing lines with the sound arm cause movements of a similar kind, but less perfect, on the paralyzed side.

Paralysis or akinesis is a loss of motor power. Monoplegia is a condition in which one limb is paralyzed; hemiplegia one in which one-half the body is paralyzed; and paraplegia one in which the two lower limbs are affected. Sometimes a double hemiplegia or diplegia occurs.

The term paralysis is sometimes used to indicate loss of any kind of function, as paralysis of sensation or secretion.

Paresis is a term used to indicate a partial paralysis. It is not

to be confounded with the term general paresis, which is a form of insanity.

The Reflexes and Their Disorders.—When an impulse started in an afferent nerve reaches the spinal cord or medulla and is thence reflected at once upon an efferent nerve, it is called a simple *reflex action.* The process is an involuntary one. It ordinarily occupies one-tenth to one-twelfth of a second. The afferent nerve may be an ordinary cutaneous sensory nerve, or it may be a special nerve whose sole function is to excite reflex action. These latter nerves are called excito-reflex. This kind is principally supplied to the viscera.

FIG. 19.—DIAGRAM SHOWING THE REFLEX ARC OF SPINAL CORD. S, Sensory excito-reflex nerves; 4, motor nerve to muscle; 5, motor nerve cell.

In neurology we have to do with four kinds of reflexes:

The skin or superficial reflexes.

The tendonous or deep reflexes.

The visceral reflexes.

Idiopathic muscle reflex.

All these may be exaggerated, lessened, or absent. Further description of these reflexes will be given under the head of diagnosis.

3. *Sensory symptoms:*

Anæsthesia is a loss of tactile sensibility.

Analgesia, a loss of sensibility to pain.

Thermo-anæsthesia, a loss of sensibility to temperature. There may be loss of cold-sense or of heat-sense, or, as is usually the case, of both senses.

The term anæsthesia is often used with a general meaning to

indicate loss of all forms of sensibility. Anæsthesia in this sense is a symptom referred to the skin, bones, mucous membranes, special senses, or viscera. The muscles have two kinds of sensibility, a sensibility to pain and a special muscle sense. Anæsthesia of the pain sense of muscle is called loss of muscular sensibility or muscular analgesia. Anæsthesia of the special muscle sense is one of the factors in causing a symptom known as ataxia.

Ataxia is a symptom due to loss of the special sensibility of the muscles, articular surfaces, and tendons. This special sense informs the individual of the degree and strength of muscular movements, and by it definite and co-ordinated movements are made possible. The weight of objects and position of the limbs are also determined by it.

In *static ataxia* there is loss of the power to preserve perfectly the equilibrium when standing. It is due to the form of anæsthesia just referred to.

In *locomotor* or *motor ataxia* there is loss of power to co-ordinate the limbs properly in motion. In these conditions there is also usually a loss of power to appreciate weights or the position of the limbs. The term *muscular anæsthesia*, however, is often used to indicate these latter symptoms.

Cerebellar ataxia is a form of inco-ordination due to disease of the central organ of equilibration, viz., the cerebellum.

The anæsthesias may be incomplete, and special technical terms are sometimes used to indicate such conditions, but they are not necessary.

Hyperæsthesia is an excessive sensibility to touch, contact, and other stimuli.

Hyperalgesia is excessive sensibility to pain, and is nearly identical with tenderness.

Dysæsthesia is an abnormal sensation such as a "thrill" or feeling of discomfort produced by ordinary tactile or painful impressions.

Paræsthesia is a term applied to all the morbid general sensations except pain. The paræsthesias include such feelings as numbness, prickling, formication, flushing, burning, itching, coldness, tickling, feelings of weariness, exhaustion, various peculiar visceral sensations. Ordinarily in speaking of paræsthesiæ, however, we refer to such feelings as numbness, prickling, and creeping.

Delayed sensation is a symptom in which an appreciable time exists, usually one or more seconds, between the time of applying a stimulus and its appreciation in consciousness. Normally a tactile sensation can be felt and responded to in less than one-tenth of a second.

Transferred or *referred* or reflex sensations are those in which

the irritation is made at one point and felt at another. Thus an irritation in the stomach causes a pain felt in the forehead. The whole class of so-called reflex pains are really transferred sensations, since in reality there is no reflex action in the process, as will be seen later. *Allochiria* is a peculiar form of transferred sensation in which an irritation applied on one side of the body is referred to a corresponding point on the opposite side.

The special senses when disordered cause symptoms which are given special names. These will be described in connection with the special subjects.

4. *Trophic Disorders.*—These are called tropho-neuroses. They consist, so far as relates to neurology, chiefly of hypertrophy and atrophy of nerves, muscle, cutaneous and mucous tissues, joint degenerations, and various skin eruptions.

The tropho-neuroses, if they affect joints, are called arthropathies; if muscles, atrophies, hypertrophies, and dystrophies; or if with atrophy there is a great substitution of fat the condition is known as lipomatosis. When nerves are affected there results degeneration.

Tropho-neuroses of the skin produce various symptoms such as herpes, pemphigus, and other eruptions, pigmentation, leucoderma, alopecia, and bed-sores.

5. *Vasomotor and Secretory Symptoms.*—The nerves supplying the blood-vessels and secreting glands work together and are usually disordered together. Separate disturbances of the vessels and glands, however, occur.

Angio-neurosis is the term given to disorders of the vasomotor centre and nerves. *Angio-spasm* is a condition in which there is increase of vasomotor tone and spasmodic contraction of the muscular coats of the arteries. *Angio-paralysis* represents the opposite condition. Such disorders affecting the skin are shown by pallor and coolness or by flushing and heat. *Angio-ataxia* is a condition of variability and irregularity in the tonus of the blood-vessels.

6. The *secretory neuroses* affect oftenest the skin.

Hyperidrosis is an excessive sweating. *Anidrosis* is excessive dryness. *Paridrosis* is a perversion of secretion in which peculiar odors or colors are noted. *Hæmidrosis* is the term applied to bloody sweating.

The secretions of the internal organs are controlled by nervous influences, but their special disturbances do not require description here.

CHAPTER III.

DIAGNOSIS AND METHODS OF EXAMINATION.

The diagnosis of a nervous disease may be simply a clinical one; that is to say, one may recognize it as belonging to a certain known and definite group of symptoms. Thus in recognizing the phenomena of epilepsy, one makes a clinical diagnosis. In other cases, and especially in all organic nervous diseases, the physician must make in addition a local, and then a pathological diagnosis. That is, we must determine the seat and nature of the disease.

A diagnosis is made by first getting all the obtainable facts in the patient's past history, then by learning from him all his subjective symptoms, and finally by making an examination according to the technical methods to be here described. In examining a patient, it is imperative that a careful search for diseases outside the nervous system first be undertaken. Then the morbid nervous phenomena should be investigated. The physician should make it an invariable rule to make this examination in a certain fixed and systematic manner. The best method is first to get the family and personal history, and then to go over the mental, cerebral, and special nervous functions serially in the way indicated under the description of general symptoms, thus: Examine—

1. Physiognomy, general condition of nutrition, complexion, physical defects (stigmata of degeneration), gait, station, posture, speech.
2. Mental and cerebral symptoms.
3. Motor and muscular symptoms, including muscular and joint atrophies, electrical reactions, and the reflexes.
4. Sensory symptoms, general and special.
5. Vasomotor, trophic and secretory.
6. Visceral centres.

In investigating the family history, it is often necessary to make very direct and probing inquiries, for patients are, as a rule, inclined to forget or ignore the existence of nervous and mental disease among relatives. The existence of consumption and inebriety, epilepsy and syphilis, in the direct line are very important facts; so also are those concerning birth. The patient should be questioned closely as to his previous diseases, especially syphilis; also as to his habits in relation to sexual indulgence, indulgence in alcohol, and smoking. In women,

the tea habit should be inquired into. The patient may be allowed to tell his own story first. Proper queries should be put to supplement this, and finally the patient should be asked to state those symptoms which to his mind are main and dominant.

We will now go over the above points in detail.

I. The physiognomy, complexion, posture, and gait being noted, the physician looks next for evidences of imperfect physical development. These are known as the stigmata or marks of degeneration.

Marks of Degeneration.—*General.*—Shortness of height, general feebleness of constitution.

Left arm and leg larger than right, long fingers.

Cranial.—Asymmetry of skull, and especially of frontal bone.

Short parietal arc (noted in epilepsy), short frontal arc (noted in petit mal).

Peculiar type of skull, *e.g.*, trigonal, scaphocephalic, plagiocephalic.

High, prominent forehead in women.

Heavy jaws, lemurian hypophesis, prognathism.

Large frontal sinuses, small orbit.

High and narrow palate (noted in idiots).

Muscular.—Unequal innervation of facial muscles on the two sides.

Depression above glabella due to over-action of corrugators (noted in epilepsy).

Squints and astigmatism.

Other Marks.—Deviation of nose; ears long, or prominent, or set too far back; absence of helix, anti-helix, or lobule.

Badly set and badly nourished teeth.

Crown of scalp double or out of place.

Small or deformed genitals, atrophic uterus.

Physiological marks are those of deficient vital activity of the organic functions, such as weak heart, low arterial tension, unstable vasomotor innervation causing coldness and flushing of extremities, weak digestion, constipation, amenorrhœa, sexual weakness or perversion.

Psychical marks are those in general of feeble or erratic mental activity, unstable will, deficient moral sense, and lack of emotional control.

While many of the foregoing stigmata have no significance in themselves, yet a combination such as impresses the observer with its preponderance is of great importance, for neuroses or psychoses developed among this class have a much more unfavorable prognosis. It is especially among neurasthenics, epileptics, severe forms of hysteria, and in the insanities that these signs are to be looked for and studied. Among normal men about two per cent have the stigmata of degeneration; among lunatics, criminals, abortive types of paranoia and primary forms of neurasthenia, the percentage is about thirty.

The accompanying table will be of help in making the investigations relating to the cranium:

Table of Cranial Measurements, in Centimetres.

	Adults.		Physiological Variation.	New-born.		End of 1st yr.		1st to 7th yr.		10th year.		7th to 18th years.	18th to 24th years.	End of 12th yr.	
	M.	F.		M.	F.	M.	F.	M.	F.	M.	F.				
1. Greatest circumference	52.0	50.0	48.5 to 57.4	34.0	34.0	42.0	42.0	34 to 46	....	49	47	46.0 to 49.5	49.5 to 52.25	....	Taken around glabella and maximal occipital point.
2. Binauricular arc	32.0	31.0	28.4 to 35.0	20.0	20.0	25.5	25.0	.....	27	...	....	.....	.....	30	Measured from *b* over through bregma to *b*, or opposite ext. aud. meatus.
3. Volume	1,500	1,300	1,201 to 1,751	385 to 450	....	700 to 1,000	....	.....	....	1,300	...	.....	.....	....	} Benedict and } Huschke.
4. Naso-occipital arc	32.0	31.0	28 to 38	22.0	22.0	28.0	28.0	.....	....	.....	...	....	.....	....	*n β t.*
5. Naso-bregmatic arc	12.5	12.0	10.9 to 14.9	7.7	7.7	....	10.0	....	....	....	12	.....	.....	....	*n β.*
6. Bregmat. lamb. arc	12.5	11.9	9.1 to 14.4	9.0	9.0	.....	10.0	.....	....	12	....	.....	.....	....	*β* to *a.*
7. Lamb. occipital arc	7.0	7.1	.....	.....	.....	.....	...	.....	....	.....	.....	.....	.....	....	*a* to *t.*
8. Antero-posterior diameter	17.7	17.2	16.5 to 19.0	...	.....	.....	.....	.....	....	.....	.....	.....	.....	....	*s* to *o.*
9. Greatest transverse diameter	14.6	14.0	13 to 16.5												
10. Cephalic or length-breadth index	82.2	83.8	76.1 to 87												
12. Facial length	12.37	.....	10.5 to 14.4	.....	....	.....	....	.....	....	....	...	.....	.....	....	From *n* to lowest point of chin.
13. Empirical greatest height	13.3	12.3	11.5 to 15	..	..	.....	..	.....	.	.....	.....	.....	.....	....	The empirical greatest height, *b β*, is obtained by measuring the sides of the triangle *m β t.*
Dolichocephaly	.....	.....	70.0 to 74.9												
Mesocephaly	.....	.....	75.0 to 79.9												
Brachycephaly	....	....	80.0 to 84.9												
Eurycephaly	.....	.....	over 100 to 150												

To understand it, it is necessary to describe the skull landmarks, and to give briefly the classification and terms used by anthropologists and alienists in describing the dimensions and shape of the skull.

Dimensions and Shape of Skull.—*General Classification.*—Anthropologists make a general classification of skulls into—

The dolichocephalic, in which the antero-posterior diameter is to the transverse as 100 is to 75 or less; the brachycephalic, in which the length is to the breadth as 100 is to 80 or more; the mesocephalic, in which the length is to the breadth as 100 is to 75 to 80. The physiological limits of variation in the ratio of length to breadth are from 100 to 70 to 100 to 90. The dimensions and shape of the skull vary with age, sex, individual, race, and with certain pathological conditions and artificial deformities. In general, however, the variations in the shape and size of the

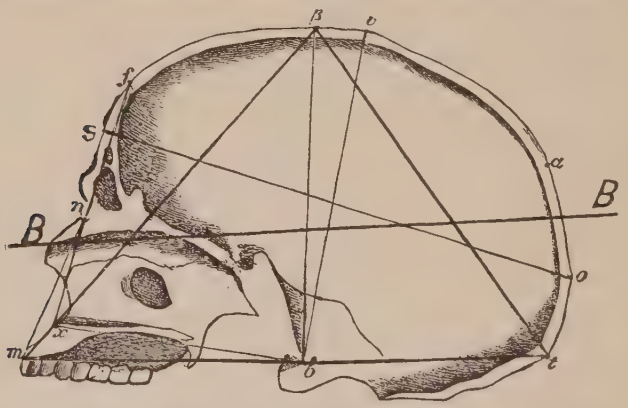


Fig. 20.—B,B, Broca's visual plane; *m*,*β*,*t*, the triangle for ascertaining the empirical greatest height (Benedict).

skulls of healthy adults of European and American races are fairly uniform.

Variations Dependent upon Age.—The proportions of the skull change most considerably in the first year, and continue to change up to the fourth year. After that, modifications are slight in amount and appear more slowly. By the end of the seventh year the skull has nearly reached its full size (see table), more nearly in girls than in boys. The chief measurements during childhood are given in the table. The protuberances and ridges are less marked in children.

The female skull is larger posteriorly, is broader, lower, with higher orbital diameter; often it has no glabella, no super-glabellar depression, and is less well marked as to its ridges, prominences, and sutures.

Variations as Regards Race.—The length-breadth index and other cranial indices and the volume are the only racial differences so far extensively studied. Even these are too indefinite factors to be of any practical value. In general, we may say that

the dolichocephalic or long-headed are: the English, Irish, Scandinavians, negroes, 73; Arabs, 74; Chinese, 76. The brachycephalic or broad-headed are: the Germans, 81; Russians, Turks, 81. The mesocephalic or medium-shaped heads are: the American Indians, 79; Hollanders, Parisians, 79.

The Variations Dependent upon Artificial Deformities, Accidents, Perversions of Growth and Development, and upon Disease.—There are certain more or less pathological variations in the shape of the skull, due to a premature ossification of a suture, or arrest of development in a centre of ossification, or to a hyperplasia, or aplasia of a part of the skull or of its contents. When one part is shut off from its natural expansion, other parts, as a rule, undergo compensatory development. This principle underlies the pathology of cranial deformities. Those deformities which it would be well to look for are—

The triangular or trigonocephalic skull. The keel-shaped or scaphocephalic skull. The acrocephalic skull. The oxycephalic or steeple-shaped skull. The flat-headed skull. The plagiocephalic or obliquely deformed skull.

Variations in the Insane, Criminal, Epileptic, and other Psychopathic Classes Generally.—Minimal measurements of the frontal arc ($n\ \beta$) are oftener found in insane and criminals. The parietal arc is also said to be often shorter in the insane, defective, and delinquent classes and in epileptics. In these classes, however, maximal excesses also often occur.

There is, as yet, no acknowledged special variation or type which is, of itself, an indication of a peculiar mental trait or specific pathological condition, although Lombroso and his followers have tried to make out such. On the whole, it is found that pronounced deformities occur often in healthy persons, while in psychopathic classes and criminals there is a large proportion of slight variations generally minimal, *i.e.*, below the mean measurements from the normal type.

For all ordinary purposes the only instruments needed in examining the cranium are a tape, a strip of lead to use as a conformateur, and a pelvimeter.

2. Investigation of Symptoms of Disordered Motility.—The recognition of gross disturbances such as hemiplegia and paraplegia is easy. To determine the exact extent of the paralysis, the patient must be made to move the limbs and contract the muscles in every possible way. The degree of paralysis in some groups of muscles can be measured by dynamometers. The ordinary hand dynamometer of Mathieu measures the degree of paralysis in the flexors. It should be graduated accurately in pounds or kilograms. I have devised an instrument by which the strength of the extensors of the hand and fingers can be tested.

The average power of pressure on the Mathieu dynamometer is, for an adult, forty to fifty kilograms for the right hand, and three to five kilograms less for the left. A woman has about two-thirds of the power of a man.

I have had constructed an apparatus by which the strength of the leg-push, *i.e.*, of the extensors of the leg, and foot and the

extensors of the thigh is tested. The anterior tibial and calf muscles can also be tested by means of an instrument called the pedometer devised by the late Dr. William R. Birdsall. A good idea of the degree of paralysis can be got by making the patient take the physician's two hands with his own and squeeze each at the same time. A malingerer or hysteric will often in this way unconsciously press much harder than he is aware. The physician's own ingenuity will suggest various ways of testing the strength of the leg and thigh muscles, such as making the patient rise on one toe, climb upon a chair, push against an object with his foot, etc.

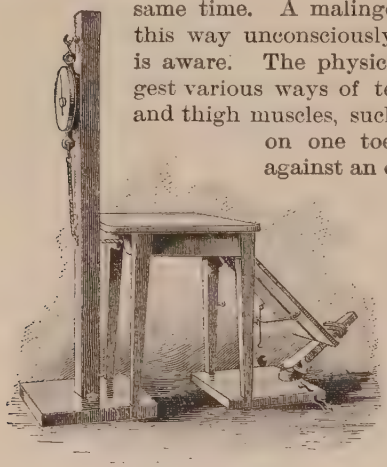


FIG. 21.—LEG DYNAMOMETER.

FIG. 22.—FOOT DYNAMOMETER.

Tremor is tested by making the patient hold out the hands and arms at full length, spreading out the fingers at the same time. To determine whether the tremor increases on volitional movement, give the patient a full glass of water, let him hold it out for a moment, then bring it to his mouth slowly. If the

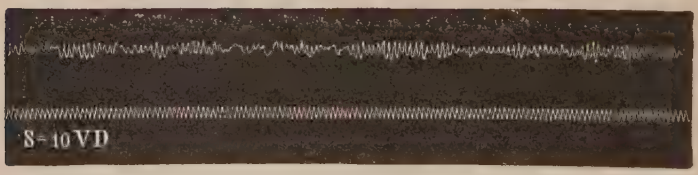


FIG. 23.—DIAGRAM OF A FINE VIBRATORY TREMOR. Ten of the divisions on the lower line equal a second.

tremor increases with this movement it is called "intentional." As a general rule, the tremor of organic disease is increased by volitional movement, and ceases during rest of the extremity. Functional tremors are usually continuous.

In some forms of tremor the hand and arm shake as a whole; such tremors are called vibratory. In other forms the tremor in-

volves only the fingers or hand. Such tremors are called oscillatory or segmental.

The apparent vibration of a tremor may range from five or six to ten or twelve per second. To determine accurately whether the tremor is *fine* or *coarse* special apparatus is needed; but one can with a little experience determine this fairly well by observation alone. Or we can use a sphygmograph as shown by Dr. F. Peterson. This instrument is fixed firmly on the table, and the tremulous forefinger held lightly against the lever. Tremor that is hardly observable by the eye can be felt by placing one's hand against the extended fingers of the patient. Tremor of the tongue and lips and facial muscles must be carefully looked for. It is tested by making the patient close the eyes tightly and show the teeth or protrude the tongue. Tremor of the whole head due to the neck muscles must be distinguished from secondary shaking of the head due to a tremor of the trunk.

Fibrillary tremor, which involves only certain fibres of the muscle, is seen oftenest in the tongue and face muscles of the extremities.

Choreic movements, tics, associated and forced movements, and the other forms of motor disturbance can be recognized by simple observation.

Myoidema is a tonic spasm of a part of a muscle near its tendonous attachment. It is produced by a sharp blow upon the muscle near its tendonous insertion. This causes the muscular fibres to bunch up into a small tumor for several seconds. Its presence indicates rapid muscular wasting from exhausting disease.

Idiopathic muscular spasm is a phenomenon of a similar nature. When the belly of a muscle is struck with a dull instrument, a welt of contracted muscle appears and lasts several seconds. It indicates an exaggerated muscular irritability.

The Examination of the Reflexes.—These, as already stated, are of four kinds: (1) the superficial or skin, (2) the deep or tendonous, (3) the visceral, and (4) the muscle reflexes.

1. A *skin* or *superficial reflex* is produced by scratching, tickling, pinching, or irritating the skin, with hot, cold, or chemical irritants. The result is a contraction of the muscles supplying the parts near the irritation. The skin reflexes which can be ordinarily brought out are the plantar, cremasteric, epigastric, abdominal, erector spinal, interscapular, palmar, and certain cranial reflexes.

The plantar reflex is produced by tickling or scratching the soles of the feet. This causes a quick, involuntary jerking up of the foot and leg.

The cremasteric reflex is brought out by scratching the inner side of the thigh or the skin over Scarpa's triangle. It causes a drawing up of the testicle on the same side.

The abdominal reflex consists of a contraction of the abdominal muscles, caused by irritating the side of the abdomen.

The epigastric reflex consists of a contraction of the upper fibres of the rectus, caused by irritating the skin of the lower part and side of the thorax.

The erector-spinæl reflex consists of a contraction of some of the fibres of the erector spinæl, caused by irritating the skin along its outer edge.

The scapular reflex consists of a contraction of some of the scapular muscles, caused by irritating the skin over them.

The palmar reflex is produced by irritating the palms of the hands.

The cranial reflexes are the lid reflex, caused by irritations of the conjunctiva or of the retina; the pupillary-skin reflex, which consists of a dilatation of the pupil caused by scratching the skin of the cheek or chin.

The levels of the spinal cord through which the impulse travels are indicated in the accompanying table:

TABLE SHOWING THE NERVES AND SPINAL CORD LEVELS BROUGHT INTO PLAY IN THE DIFFERENT SUPERFICIAL REFLEXES.

Spinal Nerve.	Superficial Reflex.	Spinal Nerve.	Superficial Reflex.
Cervical 1		*Dorsal* 8	
2		9	
3		10	Abdominal
4		11	
5		12	
6		1	
7	Scapular	2	Cremasteric
8		*Lumbar* 3	
1		4	*Knee jerk*
2		5	Gluteal
3		1	*Ankle clonus*
Dorsal 4		*Sacral* 2	Plantar
5		3	
6	Epigastric	4	
7		5	

The superficial reflexes that can ordinarily be brought out in healthy persons are the plantar, cremasteric, epigastric, and the cranial reflexes. The palmar reflex is rarely present in healthy people except during sleep, and in children. The superficial reflexes depend upon the integrity of the reflex spinal arc, and to a less extent upon the degree of cerebral inhibition. When present, they show that the spinal cord at the level through which the impulses travel is healthy. When absent, they do not necessarily indicate much of anything. For they vary in amount in different persons and at different ages. In cerebral hemiplegia during and for a time after the acute attack, they are generally lessened or absent on the affected side. Later they may be exaggerated.

The *deep reflexes* are sometimes called *tendon reflexes*. They are brought out by striking a stretched tendon or muscle or even by tapping a bone or striking the muscular fascia. The deep reflex in these cases is probably a true spinal reflex, though some assert that it is due to the direct effect of the concussion or sudden stretching upon the muscle itself (Gowers), which is in a condition of slight tonus. Either view involves the integrity of a reflex arc.

The deep reflexes are—

The patella-tendon reflex or knee jerk. The ankle reflex or ankle clonus. The wrist reflex. The triceps-tendon reflex or elbow jerk. The jaw reflex or chin jerk. The light (or pupillary) and accommodation (or ciliary) reflexes.

The *patella reflex* or knee jerk consists of a sudden contraction of the quadruceps femoris, vastus internus, and subcrureus caused by striking the patella tendon when the leg hangs loosely at right angles with the thigh. This reflex may also often be produced by striking the lower part of the muscle itself. The activity of this reflex is increased if, at the same time that the blow is struck, a voluntary contraction of some other muscles is made by the patient. Usually the patient is told to pull on his clasped fingers, or tightly shut the hands. This process is called the *reenforcement* of the knee jerk (see Fig. 24).

The knee jerk is present practically in all healthy persons except the aged. Its absence is always of significance. The nerve roots involved are those, in man, of the fourth lumbar pair. The peripheral nerve is the anterior crural. The most essential muscle is the vastus internus (Sherrington).

Ankle clonus is caused by having the seated patient extend the limb and hold it rather firmly in a semi-flexed condition. The physician takes the foot by the toe and heel and quickly flexes the foot on the leg. He thus suddenly stretches the calf muscles, and they undergo rhythmical contraction. This phenomenon does not occur in healthy people. An ankle-tendon re-

flex, however, is brought out in normal conditions by striking the Achilles tendon when the foot is held slightly flexed by the physician.

FIG. 24.—GETTING THE KNEE JERK BY RE-ENFORCEMENT.

The "paradoxical contraction" is a name given to the tonic contraction of the anterior tibial muscles caused by the physician's suddenly flexing the foot on the leg, thus shortening these muscles. This is a rare phenomenon and never found in health. The wrist reflex is brought out by striking the wrist tendons while the forearm is supinated and held limply on the hand of the physician. The triceps reflex or elbow jerk is brought out by striking the triceps tendon while the arm is sup-

FIG. 25.—GETTING THE ELBOW JERK.

ported and the forearm allowed to hang down loosely at right angles to the arm. These reflexes occur in normal individuals. The jaw reflex or jaw jerk is brought out by having the patient open the mouth and leave the jaw relaxed. A flat instrument like a paper cutter is then laid on the teeth of the lower jaw, and if this is struck smartly the elevators of the jaw contract.

The light reflex is caused by throwing a bright light into the eye, and the ciliary or accommodation reflex by making the patient look at a distant and then at a near object. The pupil

normally dilates in the former case and contracts in the latter. When the light reflex is lost while the accommodation reflex remains, the condition is called the *Argyll-Robertson pupil.*

The deep reflexes which do not occur in healthy individuals are: ankle clonus, paradoxical contraction, jaw clonus, and usually the wrist reflex. The deep reflexes may be brought out by simply striking the tendon with the side of the hand or the back of a book. For careful work, a percussion hammer is very useful.

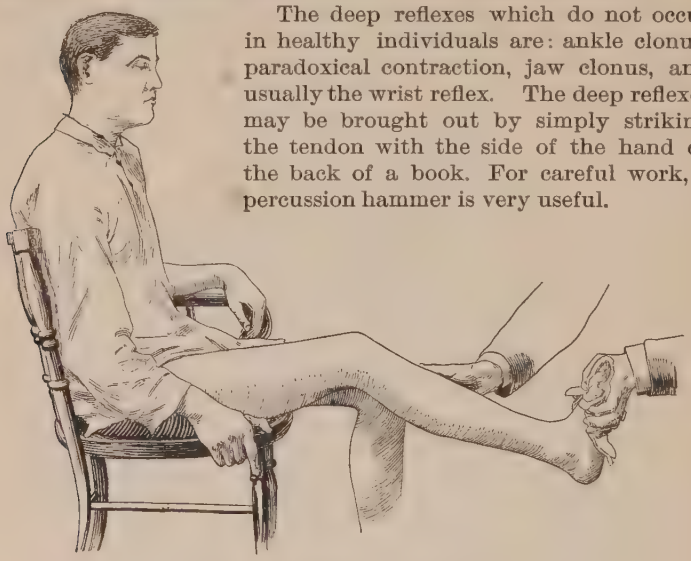


FIG. 26.—GETTING ANKLE CLONUS.

The Electrical Conditions in Disturbances of Motility.—These cannot be understood without some description of the methods of using electricity, and hence the technique of electrical examinations for purposes of diagnosis will be described under the head of Treatment.

3. EXAMINATION OF THE DISORDERS OF SENSATION.—It should be remembered that sensations are of two kinds, general or common and special.

A common sensation is one which is referred to the body. A special sensation is one which is referred to the external world, and in particular to the object which causes the stimulus. The pain from a knife cut is referred to the body, and is a common sensation. The coldness felt when a knife blade is laid on the skin is referred to the knife, and is a special sensation.

The sensory functions to be examined are:

The cutaneous.

The muscular, articular, and tendonous.

The visual, auditory, olfactory, gustatory, and space senses.

Visceral and general bodily sensations.

The *cutaneous sensations* are: (1) The tactile sense, which in-

cludes pressure and contact; (2) the temperature sense, which includes the heat sense and cold sense; (3) the pain sense. The first two are special senses, the last is a general sense.*

To test the tactile sense, blindfold the patient and use the æsthesiometer. This is an instrument with two rather blunt points, which can be separated or approximated. A hairpin or two ordinary pins can be used in its stead. Its use depends upon the fact that the power to appreciate the contact of two points on the skin gradually approximated varies with the tactile sensibility of the patient. The tongue, finger tips, and lips are the most sensitive points. The back, arms, and thighs the least sensitive.

The following table shows the average distance at which two points are appreciated as such by an intelligent adult:

Tip of tongue,	1 mm. ($\frac{1}{25}$ in.)	Tip of toes, cheeks, eyelids,	12 mm.
Tip of fingers,	2 "	Temple,	13 "
Lips,	3 "	Back of hands,	30 "
Dorsal surface of fingers,	6 "	Neck,	35 "
Tip of nose,	8 "	Forearm, leg, back of foot,	40 "
Forearm,	9 "	Back,	60–80 "
		Arm and thigh,	80 "

The figures vary somewhat with the thickness or softness of the skin and with the dulness or keenness of the nervous organization. If the distances are double those given above, it may be considered in most cases abnormal.

The tactile sense may also be tested by the writing method (Rumpf). Figures or letters are written upon the skin with a hard-pointed instrument, and the patient asked to tell them. The figures drawn are made larger or smaller in accordance with the decrease or increase of sensibility. The following table shows the different sizes as appreciated on the normal skin:

Finger tips,	0.5 cm.	($\frac{1}{5}$ in.)	high
Palm,	1 "	($\frac{2}{5}$ in.)	"
Neck,	1 "	"	"
Cheeks,	1 "	"	"
Forehead,	1 "	"	"
Arm, forearm, and back of hand,	1.5 to 2.5 "	($\frac{3}{5}$ to $\frac{4}{5}$ in.)	"
Scapula,	1.5 to 2.5 "	"	"
Calf and sole,	3 "	($1\frac{1}{5}$ in.)	"

* Psychologists deny the independence of the pain sense and assert that it is only a quality or modification of other senses.

The sense of contact which is a form of tactile sense is tested by drawing one's finger or a bit of cotton lightly over the skin. The sense of locality or power to localize a point on the skin that has been touched varies with the tactile sense and with the muscular sense. It is tested by placing the finger lightly on a given spot and telling the patient with closed eyes to place his finger on the part touched. He should come within 5 cm. of it. In slight degrees of anæsthesia dependent upon disease of the sensori-motor areas of the cortex of the brain, this is an important test. Further tests may be made by moving points along the skin and asking the patient to indicate the direction of the motion; or by laying various shaped objects on the skin and asking the patient to tell their shape or position. Still another method is recommended by Oppenheim. It consists in testing at the same time with the æsthesiometer symmetrically situated parts of the skin on the two sides of the body.

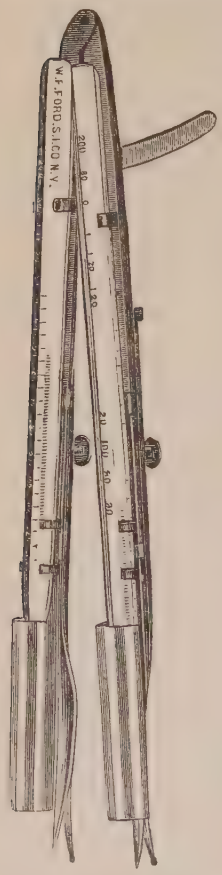


FIG. 27.—COMBINATION ÆSTHESIOMETER.

To test the *pressure sense*, use, if desired, the bar æsthesiometer. A more convenient way is to have the patient rest the hands on a table and then try and determine the weight of different objects. The lightest weight that can be appreciated on the hands or face is one of about 0.02 gram (gr. $\frac{1}{3}$). Differences of light weights of 1 and 5 grams and of 25 and 30 grams are about all that can be ordinarily appreciated by the skin. Much smaller differences, of 0.5 to 2 grams, can be detected if great care is used. Weighted rubber balls may be used in the foregoing test. I prefer to use differently weighted metal bodies, held by a wire. Pressure sense is acute on the forearm and abdomen, where locality sense is feeble; also on the brow, temples, and back of the hand.

Most of the above tests are not ordinarily needed. With two pins, using the heads, the presence and degree of anæsthesia can be detected and approximately measured.

The *temperature sense* is tested by test-tubes filled with hot and cold water, or by using hot and cold spoons, or roughly by breathing and then blowing on the part. A thermo-æsthesiometer may be used. This has a round, flat surface 1 cm. in diame-

ter, and contains in its terminals thermometres by which the degree and differences in temperature may be noted (Fig. 27). A small heated or chilled surface is appreciated much less easily than a large one. The indifferent range where objects are felt to be neither warm nor cold is from 27° to 30° C. (80.6° to 86° F.). Fine differences (0.2° to 1.5° C.) are appreciated above the indifferent range. Lower down in the scale, differences from 1° to 1.3° C. (2° to 3° F.) are appreciable. It may be considered a morbid symptom if temperatures of 60° to 65° F. are not felt as cold, or temperatures of 86° to 95° F. are not felt as warm; also if between the ranges of 1° C. (32° F.) and 40° C. (104° F.) differences of 2° C. are not appreciated.

A painful degree of sensitiveness to heat or cold sometimes exists. These conditions are called hyper-thermalgesia and hypercyalgesia (Skinner, Starr). When the heat or cold is intense, a sensation of pain is felt. Cold-pain is produced more easily in some places, such as the elbow, than others, as, for example, the finger tips. Cold-pain is produced by temperatures of from +2.8° C. to −11.4° C. Heat-pain is produced by temperatures of from 36.3° C. to 52.6° C.

The *pain sense* is tested by pricking the skin with needles or the sharp points of an æsthesiometer. The faradic battery with metal points or a wire brush may also be used. Instruments for pinching the skin and measuring the sensibility by the strength of the pinch have been devised. The power of localizing pain is lessened in proportion to the analgesia.

The *muscular sensibility*, *i.e.*, the general or pain sensation of muscles, is tested by passing the faradic current through the part.

Pain and temperature sense are usually affected together.

Delayed Sensation.—The time taken for a sensation to be felt and produce a voluntary response is, for—

A touch on the hand, about . . .	0.12 second
" " " foot, " . . .	0.17 "
Hearing, ...	0.13 "
Sight, ...	0.16 "
Taste, ...	0.15 "

The tactile sense, as well as the other special and the general sensations, may show a delay in conduction. The tactile sense especially should be tested on this point. The delay may amount to several seconds.

Double Sensations or Polyæsthesia.—When the touch of one point is felt as two or more, the symptom is known as polyæsthesia.

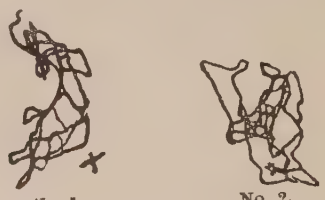
Referred sensations and *allochiria* are described under Symptomatology.

Tests for the Condition of the Special Sense of Muscles, Joints, and Tendons, i.e., for Ataxia.—Anæsthesia of the special sensory nerves of the muscles, joints, and tendons causes ataxia and inco-ordination. Muscle anæsthesia causes chiefly a loss of *weight sense* or loss of power to determine weights. It is tested by the use of weights suspended by a string so as to exclude pressure sense; also by causing the patient to squeeze a dynamometer up to a certain fixed number.

Fig. 28.—Ataxiagraph.

In articular and tendonous anæsthesia there is loss of posture sense. It is tested by the physician's moving the patient's limbs and having the blindfolded patient tell in what direction the movement is made. Or he is told to follow with one limb the movements which the examiner makes with the other.

Muscular, articular, and tendonous anæsthesia usually exist together; there is then a combined ataxia. Combined ataxia shows itself in standing and in locomotion and other voluntary movements. Thus we have a *static* ataxia and *locomotor* or motor ataxia. Static ataxia or inability to stand (or sit) without swaying or irregular move-



No. 1. No. 2.

Fig. 29.—Ataxiagrams. No. 1, Made with eyes open; No. 2, made with eyes closed.

ments is tested by making the patient stand with the eyes closed and the heels and toes close together.

Normally, the head moves not over an inch in this position, and the patient holds the head and body more rigid with the eyes closed than with them opened. In ataxic states the reverse is true, and decided swaying or even complete loss of equilibrium occurs with the eyes closed, or even with the eyes open, and the base narrowed by putting the feet together. This phenomenon is called the "Brauch-Romberg symptom." In static ataxia, muscular and articular sensations are both involved. The degree of this can be accurately measured by the ataxiagraph. With the

eyes open a healthy person standing erect with feet together tends to sway forward. The antero-posterior excursion of the head averages 3.7 cm. (1½ in.), the maximum being 9 cm. (3½ in.). The lateral excursion averages 1.9 cm. (¾ in.), maximum being 5.4 cm. With the eyes closed the antero-lateral excursion averages 3.4 cm. (1¼ in.), maximum 3½ in.; the lateral excursion 1.9 cm. (1¾ in.), maximum 6.8 cm. (2⅝ in.).

In other words, the person stands a little steadier with the eyes closed, the average excursion being 1½ × ⅜ in. with eyes open, 1¼ × ⅜ in. with eyes closed (Bullard and Brackett).

Ataxia of motion is tested by the gait. The patient cannot walk a straight line and cannot walk without watching the floor with the eyes. The arms cannot be moved in a co-ordinate way. With the eyes closed, the patient cannot place the finger on the tip of the nose, or lobe of the ear, or any indicated spot. Ataxia of motion involves especially the articular and tendonous sensations, but not these exclusively.

It may be measured by noting how close the patient keeps upon a given line ten feet long in walking; how near he can place the finger upon the centre of a board marked like a target. The patient is placed ten feet away, and made to walk directly at it and place the finger in the centre.

To sum up the foregoing, we have:

	Due to	Tested by
Ataxia	Muscular anæsthesia.....	Weights, etc.
"	Articular and tendonous anæsthesia.	Position of limbs.
"	Combined forms, *e.g.*, static ataxia...	Co-ordinate movements.
"	Locomotor ataxia.....	Station and gait.

There are, besides this, certain forms of inco-ordination which are allied to tremor, and are disorders of the sensori-motor cortical areas or efferent nerve tracts. Nor is it absolutely established that in the above-described forms of ataxia there is not an involvement of the efferent nerve mechanism.

Vision.—The special modes of examination are given under the head of Diseases of the Optic Nerve and Ocular Muscles. The special points which the neurologist must investigate are visual acuity, astigmatism, errors of refraction, limitation of the visual field, exophthalmia, retraction of the bulb, color-blindness, the state of the pupil and its reflexes.

Hearing.—The special methods of examination are given elsewhere. The points chiefly to be investigated are acuity, range, bone conduction, aerial conduction, electrical reactions.

Sense of Smell.—See p. 107.

Sense of Taste.—See p. 146.

CHAPTER IV.

THE CAUSES AND THE PATHOLOGY OF NERVOUS DISEASES.

General Etiology.

The subject of the causes of nervous diseases must be studied from two points of view.

First, we must learn the predisposing and exciting causes of nervous diseases in general. Second, we must learn the causes of the particular pathological change which constitutes the disease.

I. The causes predisposing to nervous disease relate to age, sex, condition, heredity, race, climate, civilization, occupation, habits, diatheses, infections, and poisons.

Age and Developmental Influences.—In early age, paralyses and convulsive troubles are more frequent, while sensory neuroses are rare. In adolescence, many severe constitutional neuroses and neuropathic tendencies develop, but organic disease is rare. In adult life, the inflammatory and degenerative troubles are at their height. In old age, those disorders which are dependent on disease of the blood-vessels, such as apoplexies and softening, appear.

The special nervous defects and disorders more especially associated with early developmental influences are these (Clouston):

1. Of the embryonic stage: Acephaly, genetous idiocy.
2. Of the period of most rapid brain growth—birth to seven years: Convulsions, pavor nocturnus, tubercular meningitis, meningeal hemorrhage and encephalitis, epilepsy, idiocy, infantile paralysis, neurotic fever, febrile delirium.
3. Of the period of co-ordination of motion and emotion—seven to thirteen: Chorea, epilepsy, asthma, migraine, myopathies, hereditary degenerations of the cord.
4. Puberty and adolescence—thirteen to twenty-five: Epilepsy, neurasthenia, hysteria, migraine, sexual perversions, paranoiac tendencies, hereditary myopathies and myelopathies, insanities.

Sex.—Sensory and functional disorders are more frequent in women; motor and organic disorders more frequent in men.

Condition and Occupation.—No general facts will be laid down here. Celibates, however, it may be said, suffer more from nervous disorders than married people. It will be shown later that certain occupations entail special nervous disorders, and that in-door life promotes functional nervous diseases.

Habits.—Excessive indulgence in alcohol is a most prolific cause of nervous disease, chiefly by the action of this substance on the blood-vessels and the stomach. Excesses in eating, in tea-drinking, irregularity in sleeping, and bad habits of working predispose to nervous disease. Sexual excesses are usually the result rather than the cause of nervous disorders.

Heredity.—There are few nervous diseases which are, strictly speaking, inherited. But an over-irritable, unstable, inadequately developed, and badly nourished nervous system—one with a tendency to disease—may be inherited. This is known as the *neuropathic constitution.* Excessive intellectual labor and mental strain during the time when children are being propagated tends to produce a neuropathic taint in the descendants. When both parents are themselves of a highly nervous temperament, their children are apt to be more so. The existence of one of the severe forms of constitutional nervous disease, like epilepsy or hysteria, in one parent is likely to produce neuropathic children. Intermarriage does not cause neuroses, unless father and mother are of closely alike temperaments. Excessive alcoholic indulgence in one or both parents, syphilis, injuries or fright to the mother during pregnancy, may result in neurotic children. The mother transmits neuroses and neurotic tendencies more than the father. A certain form of nervous disease may not be transmitted in the direct line, but will be found in different branches of the same family, as, for example, in uncle, nephew, and cousin. This is then called a "family disease."

Climate and Civilization.—Nervous diseases are most frequent in temperate climates, and in those which are dry and elevated. They increase with the progress of civilization and the greater strain, complexity, and luxury of modern social life. Those organic nervous diseases which are largely dependent on vascular disease are frequent in the poorer classes among whom syphilis, alcoholism, and bad feeding prevail. Functional and degenerative disorders are frequent in the higher classes. Nervous diseases, if we except those of the degenerative type, prevail more in urban populations.

Diatheses.—The rheumatic and gouty diatheses predispose to nervous troubles, more especially those which are of a peripheral and functional nature. Lithæmia, a condition in which the products of tissue waste are not properly oxidized and eliminated, has a similar influence.

Trauma and Shock.—Hemorrhages and trauma may be a direct cause or may predispose to nervous disease. Trauma and shock may cause functional diseases such as neurasthenia, or may lead to the development of insanity or indirectly to degenerative

organic disease. Mental shock, and especially a fright, oftener than severe bodily injury, leads to the development of functional neuroses. Hemorrhages, exhaustion from excessive work, and anxiety are potent causes.

Infections.—In comparison with their frequency, the infective fevers are not great factors in producing nervous disease, but practically they often play an important part. Scarlet fever is the most dangerous disorder in this respect. Measles perhaps ranks next, then follow diphtheria, typhoid fever, and pertussis. Among chronic infections syphilis ranks first; malaria, the pellagra, and beriberi are also to be mentioned.

Poisons.—Alcohol, tea, coffee, and lead, mercury, copper, arsenic, are to be placed among the frequent causes of nervous disease.

Reflex Causes.—Among other causes are local disease of viscera, such as renal, uterine, and ovarian diseases, dyspeptic and liver disorders, visual and auditory troubles.

II. The causes of particular pathological types of disease.

Inflammatory diseases of the nervous system, as of other systems, are caused almost always by microbic infection. This is brought about directly by injury and exposure to external agencies or indirectly through the blood. Inflammatory disorders occur oftenest in early and middle life, and in men oftener than in women. *Degenerative diseases* are observed oftener in middle and later life, and result from excessive nervous strain, syphilis, exposures, poverty, chemical poisons, alcohol. Heredity is also an important factor.

Diseases of the blood-vessels occur in middle and later life. Bright's disease, the gouty diathesis, alcohol, syphilis, over-feeding, and chemical poisons such as lead are among the causes.

Functional spasmodic diseases usually develop early in life when general in character, like epilepsy and chorea; but later in adult life if local, like writer's cramp and wry-neck. They often follow infective fevers. Hereditary influence is here important.

Functional sensory disorders rarely begin before the age of seven to ten, and are more common in women and in temperate climates. A certain class of them, however, like facial neuralgia, develop with the degenerative period of life.

General nutritional disorders, like neurasthenia, are more frequent in youth and middle life, in active and new civilizations, in temperate climates, especially those which are dry and cool or elevated.

Diseases of the peripheral nerves are caused by injuries, and by poisons like alcohol or infections like that of beriberi or by the auto-toxæmia of gout, rheumatism, and diabetes. They occur chiefly in early and middle life.

General Pathology.

The nervous system is composed of nerve cells, nerve fibres, connective tissue, the neuroglia, blood-vessels, and lymphatics. Its disorders involve one or more of the above tissues.

The following is a list of the forms of disease which affect the nervous system:

1. Malformations, agenesis.
2. Hyperæmia, anæmia, hæmorrhage, œdema, and arterial and venous diseases.
3. Inflammations.
4. Degeneration and atrophy, softening, sclerosis, gliosis.
5. Tuberculosis and syphilis.
6. Tumors and parasites.
7. Nutritive and functional disorders.

Inflammation.—The pathology of most of the above types of disease will be given elsewhere, and does not call for discussion here. It is, however, of the utmost importance that the student have a clear understanding of the nature of inflammation and degeneration as they affect the nervous tissue. Inflammation is a morbid process which has to deal primarily with blood-vessels, lymphatics, and connective tissue. Inflammation, teleologically, is the reaction of the organism to an irritant. Wherever there is inflammation, there is irritation. The irritant in inflammation is practically always the product of microbic action or of some irritating product of tissue change. Without some microbic or tissue irritant there can be no inflammation. We make this exception only: that certain chemical substances, such as alcohol, arsenic, and lead, may at times excite a form of inflammation which is, however, probably, primarily a degenerative or destructive process. Inflammation, when the irritant is removed, tends to subside. It is a regressive, not a progressive, process. Bearing these facts in mind, it will be found that inflammations of the nervous tissues never or very rarely occur without the presence of some microbe or some destructive process whose irritant products excite inflammatory reaction.

Inflammations may be divided into the (1) exudative and the (2) productive forms (Delafield). The exudative inflammations may be simple, without necrosis, with necrosis; purulent; purulent and necrotic.

1. *Simple exudative inflammation* is accompanied with congestion stasis, emigration of white corpuscles, and perhaps diapedesis of red cells, transudation of blood serum, and formation of fibrin, the total result being an exudate containing white blood cells, now called pus cells, and fibrin in varying proportions.

There is, in some cases, no destruction of tissue; and on subsidence of the inflammation the tissue returns to its normal condition. In other cases the inflammatory action destroys some of the nerve tissue.

In *purulent inflammation* there is great accumulation of pus cells and less relatively of fibrin.

If the tissue is destroyed, it is a *purulent and necrotic inflammation*.

In some exudative inflammations there is increase of connective tissue from the start, and the process continues till the inflammation subsides. Most exudative inflammations are acute or subacute. *Inflammatory edema* is a form of exudative inflammation.

2. *Productive* or *proliferative inflammation* is a process in which there is little congestion and exudation, while new connective tissue is slowly formed. Productive inflammation is usually chronic; tubercular and syphilitic processes are varieties of productive inflammation.

The microbes which are known to produce inflammations of nervous tissues are the ordinary pyogenic streptococcus, the diplococcus intra-cellularis, the diplococcus pneumoniæ, a bacillus like that of typhoid fever. The bacillus tuberculosis sometimes causes a purulent inflammation. The supposed rabies bacillus, the bacillus of leprosy, the anthrax bacillus, and the micro-organism of infectious multiple neuritis may be added to the list.

The principle poisons which may cause chronic productive inflammations are alcohol, lead, and arsenic.

Certain irritating auto-toxæmic agents, such as occur in gout, rheumatism, diabetes, and states of inanition, appear able at times to cause productive inflammations.

CLASSIFICATION OF INFLAMMATIONS.

Form.	Cause.	Example.
Simple exudative, with or without necrosis.	Microbic or toxic.	Meningitis. Poliomyelitis.
Purulent, with or without necrosis.	Microbic	Meningitis and encephalitis.
	Microbic	Acute purulent myelitis.
Productive or proliferative.	Microbic or toxic.	Chronic meningitis. Leprous neuritis.

DEGENERATIONS AND SCLEROSES.—By degeneration is meant a gradual death of the nerve cells and fibres, or in other words of the parenchyma of the organ. The cells swell up, become

granular and fatty, and then either break up and become absorbed or enter into a condition of a dead coagulum (coagulation-necrosis). Degenerations may be acute or chronic, primary or secondary.

Acute degeneration causes a condition known as *softening* or *necrosis*. It is due to cutting off of vascular supply, direct injury, and to necrotic and inflammatory poisons. Acute degeneration may be followed by a reparative process which is called a reparative or reactive inflammation, and which ends perhaps in producing a cicatrix or sclerosis.

Chronic degeneration is accompanied and followed by a proliferative process which results in the production of connective tissue and sclerosis.

Sclerosis is a process of connective-tissue proliferation, as a result of which the normal or injured parenchyma is supplanted by fibrous tissue. The word sclerosis is usually employed in describing degenerative diseases, though it indicates the result rather than the primary nature of the process.

A *primary degeneration* is one in which the process is due to inherent defect in nutrition or to some poison acting directly on the cell or fibre.

A *secondary degeneration* is one that is due to a cutting off of nerve fibre or cell from its trophic centre, or to an injury or shutting off of its vascular supply. Ordinarily, in speaking of secondary degenerations one refers to those due to the first-mentioned class. Practically, primary and secondary degenerations often occur in the same disease.

Degenerations.	Forms.	Examples.
Acute and Chronic	Primary	Myelomalacia, Progressive muscular atrophy, Locomotor ataxia.
	Secondary and mixed	Chronic myelitis, Secondary lateral sclerosis.

Degenerations are caused by certain poisons, such as arsenic, phosphorus, lead, and the poisons of infectious disease. Degenerations also result from obliterating arteritis such as occurs in old age or from humoral poisons. Degenerations sometimes are due apparently to an inherent defect in the cell nutrition—a premature death of it; also to causes yet unknown. The question as to whether certain scleroses are forms of productive inflammation or of chronic degeneration is one still undecided. It is probable, however, that many of the so-called chronic inflammations of the nervous system are really degenerative processes, and that the primary trouble is in the parenchyma, and not in the connective tissue.

Gliosis.—It is contended by some French pathologists (Chaslin, Déjerine) that some of the chronic degenerative diseases are the result of a proliferation of neuroglia, not of connective tissue. This process is called gliosis. Its existence is not yet satisfactorily established.

Nutritive and Functional Disorders.—A fundamental peculiarity of nervous tissue of man must here be noted.

Nerve cells once destroyed never develop again.

The same is true, though not so absolutely, for the nerve fibres running in the central nervous system.

Peripheral nerves may grow again when cut or destroyed. They always grow *from* their trophic centre. Nerve tissue in brain, cord, or periphery can never be sutured so that it will functionally unite by direct union. There are a few apparent exceptions.

A further peculiarity of nervous tissue is that it is dependent for its integrity upon two things, blood supply and trophic influences. The nerve cell is solely dependent on a proper supply of blood, and dies when this is withdrawn. But the nerve fibre is more dependent on the trophic influence of the cell to which it is connected and of which it is a prolongation. It dies when cut off from its cell, but it can get along for a time with but little direct blood supply.

Underlying nutritive and functional disorders there exist: A congenital inadequacy of the nervous system to do its work. Disturbances in blood supply. Poisonous conditions of the body, due to substances generated within or introduced from without. Reflex irritants. Exhaustive conditions from overwork.

CHAPTER V.

HYGIENE, PROPHYLAXIS, TREATMENT.

In the treatment of nervous disease, the physician attempts—1. To relieve distressing symptoms. 2. To secure radical cure. 3. To prevent return. This calls for various measures which may be classed under the heads of general hygiene, diet, exercise, climate, hydrotherapy, massage in various forms, electricity, drugs, external applications, and surgical intervention.

General Hygiene.—To secure and keep steady nerves, and to prevent the supervention of organic nervous disease, would require a considerable reconstruction of the present social system. I can only give some hints as to the kind of advice physicians should give to help along the desired end. Thus two people of very nervous temperament should not marry. Children should be brought up to eat slowly a mixed diet, to sleep early and long, to play in the open air, to learn self-control and obedience. Their parents should keep from them all infective fevers. Adults need to keep in mind but two words—moderation, exercise. With these they need not fear the use of alcohol, tobacco, tea, coffee, or even occasional irregularities in sleeping and eating. Physical and mental shocks, infective fevers, poisons, and syphilis are prolific promoters of nervous disease.

Diet.—For brain-workers in general, the best diet is a nitrogenous one, but it should contain some fat. Water should be drunk plentifully except by the obese, while the total amount of food should be less than when severe muscular exercise is taken. The best foods are meats, especially fowl; fish, eggs, milk, buttermilk, cocoa, green vegetables, and stale bread with plenty of butter. If there is a tendency to constipation, farinaceous foods and green vegetables may be made the prominent articles of diet in one of the daily meals, and stewed fruit and some alkaline water added. The drinks of brain-workers should be mainly plain and alkaline waters. Alcohol can be taken in moderation by some brain-workers without harmful results. It may even secure an increased capacity for work.

In persons of a sensitive and irritable nervous system, those who are classed popularly as "nervous," neurasthenic, or hysterical, the above rules apply as to a nitrogenous diet, plus as much

fat as can be digested. There is a class of nervous persons who of themselves find that they cannot take anything sweet without producing headaches, rheumatic pains, and dyspeptic symptoms. These persons should live on meats, fish with butter, oysters, cream and milk, cod-liver oil, and fat pork. Beef tea with the white of an egg or some peptonoids forms a very nutritious dish. It has been the canon of medicine for many years that animal food must be the soul of the neurotic's diet. Most nervous persons find in addition that green vegetables like spinach agree very well with them. Stale bread can be taken twice a day freely, plenty of butter being used upon it. The dietetic breads from which the starch has been removed are sometimes useful, but are, as a rule, unpalatable, and soon cause disgust.

When a rigid diet is to be laid down, there is no better list for nervous invalids than the following: fowl; beef; mutton and lamb; fish, boiled or broiled; oysters; milk; butter; eggs, raw or soft-boiled; cocoa; graham bread and gluten bread; spinach; Brussels sprouts; string beans; stewed fruits slightly alkalinized.

Nervous patients, especially hysterical patients, should not use alcohol at all. Tea and coffee can be taken in very small amount. The various alkaline mineral waters may be used temperately with impunity, but none of them have much specific effect in relieving nervousness or curing the nervous temperament.

Water should be drunk between or before meals and a moderate amount at meals. At least three pints of liquid should be taken daily. American neurotics do not drink water enough. They have half-desiccated nerves, and desiccation increases nervous irritability. An exclusive milk diet is indicated in some forms of hysteria, hypochondriasis, and neurasthenia accompanied with dyspepsia. Karell's method is to give four to eight ounces of warm skim-milk at 8 A.M., 12 M., 4 P.M., and 8 P.M. The amount is gradually increased.

Exercise.—As a prophylactic against nervous disorders, the value of exercise, if taken out of doors, can hardly be overestimated. Brain-workers are better for moderate exercise, but they do not need much; and after twenty-five, severe intellectual work can rarely be done by persons in athletic training. Before the age of twenty-five, when the system is exuberant with vitality, hard study and hard physical exercise can be pursued successfully together by some. Persons of a neuropathic constitution are benefited by regular exercise only when it interests the mind. In-door gymnasium exercise with the ordinary apparatus does no good except through the bath that follows it. In many forms of chronic organic nervous disease exercise is to be prohibited. These will be discussed later.

The best forms of exercise are those (1) which take one out of

doors, (2) interest the mind, and (3) call into play the muscles of the chest and arm. Walking fulfils but two and often only one of these conditions. Calisthenics are useful when they interest and are vigorously done. Horse-back riding and bicycling fulfil best the conditions required for a good form of exercise. And bicycling is the cheaper, more practicable, and generally better liked of the two. Dr. Graeme M. Hammond has called attention to the special value of cycling in nervous diseases. Lawn-tennis, badminton, golf, are all exercises which can be taken up by both sexes and at nearly all ages.

HYDROTHERAPY.—Hydrotherapy is the science of applying water in the treatment of disease. The modes by which it is used in neurological therapeutics are:

I. General hydrotherapy:
   1. Tonic hydrotherapy.
   2. Sedative hydrotherapy.
   3. Indifferent baths for mechanical purposes.

II. Local hydrotherapy.

1. *Tonic Hydrotherapy.*—For purposes of stimulating nutrition and increasing vasomotor tone we employ cold plunges, the rain bath or shower, the jet, cold sponging, cold sitz-baths, cold sheets, local applications of ice or cold compresses, or cold rubbing, ice-bags, brine baths, brief cold packs, and sea bathing.

The Cold Plunge.—The patient fills the bath-tub with water at from 60° to 70° F. He then steps in, and at once jumps out and rubs himself vigorously until reaction occurs.

The Rain Bath, Jet or Needle Bath.—The patient stands in a tub with the feet preferably in warm water, and allows the cold water to fall on the back and rest of the body for one or two minutes. Or a solid jet of cool water is thrown with force upon the back of the patient, by an attendant who stands ten or twelve feet away. The cold jet may be alternated with warm. Cold sitz-baths are taken at a temperature of 70° to 80° F. from ten to thirty minutes. The cold sheet or drip sheet is used by wringing a cotton sheet out in cold water and wrapping it suddenly about the standing patient, who is then vigorously rubbed.

Ice-bags are worn upon the spine for one or two hours once or twice, or oftener, daily; or they may be applied for one or two hours at night. Most of these measures have a general stimulating and tonic effect.

The half-bath and wash-off consists of a tub partly filled with water at a temperaure of 65° to 70° F. The water only half covers the reclining body. While lying in it, the patient is vigorously rubbed. A cold cloth may be laid on the head. After five to twenty minutes, affusions of colder water are poured over the shoulders. The bath may be made as warm as 80° at first.

Brine baths contain about two per cent of salt, this being about the amount in sea-water—twenty-five pounds to thirty gallons of water. They are given at a temperature of 100° F. for twenty to thirty minutes daily, or cool baths at a temperature of 70° F. may be given for five to ten minutes, the patient exercising meanwhile.

Physiology.—Cold applications produce a local contraction of blood-vessels, followed by dilatation. There is usually increased tissue metamorphosis, increased secretion of urine, increased absorption of oxygen, and increased excretion of carbonic acid. In non-febrile persons, cold applications abstract some heat, but they also stimulate the heat-producing centres, so that the total effect is to increase the heat of the body. Only very cold baths lessen heat production as well as excretion of CO_2. Cold baths at first accelerate and then tend to retard pulse and respiration. Cutaneous sensibility is at first increased. After a cold bath there is a sense of exhilaration and increased muscular power, provided the bath be not too cold or too long continued. The duration necessary to produce a reaction varies with different people, and some weak and sensitive patients never can be made to react. Cold baths systematically taken furnish a kind of vasomotor gymnastics. The neuro-mechanism controlling the blood-vessels becomes more supple, and the tendency to local congestion of the viscera and mucous membranes is prevented. The shower and jet furnish the most valuable means of securing tonic effects in nervous disorders. These are not used with cold water alone. It is often better to apply at first a warm stream at 95° or 105° F., and then gradually lower it, or to apply the hot and cold jets alternately. In this way tonic effects can be obtained even with very feeble persons.

2. *Sedative Hydrotherapy.*—The sedative baths are the lukewarm bath, the wet pack, Turkish and Russian baths, the hot sitz-bath, pedal baths, compresses and fomentations, and hot-water bags.

The lukewarm bath is given at a temperature of 95° to 98° F. for ten minutes to half an hour daily. If a slight tonic effect is desired also, the patient should receive an affusion afterward, *i.e.*, basins of cool water at 60° to 70° F. should be poured over the shoulders. The addition of salt or of pine-needle extract is often useful.

The Wet Pack.—A large, thick blanket is spread upon the bed, and upon this is laid a linen or cotton sheet wrung out in cold water, 40° to 60° F. The nude patient lies on this, and the sheet is then smoothly wrapped about him, the head and feet not being included. The sheet is carried between the legs and made to lie evenly in contact with the body. Then the blankets are

folded over him, and other blankets may be piled upon this. Sometimes it is well to place hot-water bottles at the feet and a cool compress on the head. The patient lies in this pack for thirty to forty-five minutes and is then rubbed off. A cool affusion may be given first. In delicate patients it is well at first to simply wrap the patient in warm flannels until free perspiration results. Then give a cold affusion or wash-off and rubbing.

In *Turkish baths* the patient is exposed to a temperature at first of 130° or 140° F. for fifteen to thirty minutes, and then to one of 200° F. or 250° F. for a shorter time. This is followed by massage and a cold affusion or plunge or shower. The effects of these baths are somewhat tonic if not too prolonged. The patient should never go into the hotter rooms until he perspires, and he should select bath-rooms that are well ventilated. *Russian baths* have similar effects, but the bodily temperature is raised to a higher degree in them than in Turkish, owing to the lessened amount of perspiration.

Hot Sitz-Baths.—The patient sits in water at a temperature of 100° to 125° F. for twenty or thirty minutes. Sometimes mustard is added. *Hot compresses* consist of layers of flannels wrung out in hot water and covered with dry flannels and rubber cloth. They are used to relieve local pains and inflammations. They may be applied over the abdomen for insomnia. *Hot sprays and douches* are used for similar purposes as fomentations. The hot spinal bag is applied at a temperature not above 120° F.

Physiology.—Warm baths, if applied in the form of the moist pack, followed by sponging with tepid water, lessen temperature by increasing heat radiation and conduction. If applied so as to prevent radiation, the bodily heat is raised. Warm baths increase the circulation of the skin, lessen cutaneous sensibility, withdraw blood from the central organs, increase the exhalation of CO_2, but lessen the respiratory activity on the whole. Nitrogenous metabolism is increased from two to three per cent, and more urea is excreted. Pulse and respiration are increased. Nervous excitement is lessened, and the general effect is to cause sedation and a feeling of languor. The wet pack is a most useful sedative in neurasthenia and insomnia, and may take the place of medicinal sedatives, like the bromides. It should be given three or four times weekly, or for a short time daily. The lukewarm bath ranks next in its sedative efficacy. It is believed that applications of water to the feet and abdomen especially affect the intra-cranial circulation; given to the thighs and wrists, the pulmonary circulation; cold causing congestion, and heat anæmia, of the distant parts. Cold to the spine is believed to cause at first constriction, and later dilatation, of the thoracic, abdominal, and pelvic viscera; heat has the opposite effect. Hence cold

applications are used to relieve cold feet and anæmic conditions of the viscera.

The fact must be borne in mind that cold baths and frequent bathing of any kind debilitate some few persons.

Massage.—The term massage may be made to include all the manipulations of the body for the purpose of curing disease. The different methods of applying it as classified by Jacoby are:

Effleurage or gentle stroking. The maximum force to be applied here should not exceed the weight of the hand. *Massage à friction* or rubbing. Energetic strokes with one hand and strong circular or to-and-fro friction with the other. *Pétrissage* or kneading. *Tapotement* or percussion with the fingers, hands, or instruments. *Functional movements*, passive, active, and combined with movements made by the operator.

The physician may be reminded that a male operator is a *masseur*, a female a *masseuse*, and that the patient is *massèd*.

Massage accelerates the lymph and venous currents, and thus promotes absorption. It increases the rapidity and force of the heart-beat, except abdominal massage, which slows the heart and helps to relieve local congestions and inflammatory deposits. It presses and stretches the terminal nerve filaments, increases the irritability of motor nerves and the contractility of muscles. It may either increase or lessen the irritability of sensory nerves according as it is applied. Of the various forms of massage, *tapotement* is the most frequently useful, and is the kind almost exclusively used in neuralgias. It is applied not only with the fingers and hand, but also by the aid of rubber tubes known as muscle beaters, rubber balls with rattan or whalebone handles, percussion hammers and various percuteurs, such as Granville's and Jacoby's.

Massage is of considerable value in certain forms of atonic neurasthenia and hysteria, associated with anæmia, dyspepsia, and feeble circulation, in hemiplegia, in the paralyses of peripheral origin, in functional spasm, especially in some forms of writer's cramp and allied neuroses, in cerebral hyperæmia, insomnia, constipation, and in headache, and some neuralgias, especially those about the head, neck, and arm. It is contraindicated in heart disease, arteritis, or where there is danger of dislodging a thrombus.

Climate in Nervous Diseases.—The factors which make up a special kind of climate are:

1. Purity of air; 2, temperature; 3, humidity; 4, sunlight; 5, rarefaction of the air; 6, ozone; 7, wind; 8, electricity; 9, soil; 10, trees; 11, social conditions.

Regarding these points, some facts are very well settled. The air in the country is purer than in cities. The air on the sea and

at high levels is purer than in other localities. The temperature above the sea-level diminishes about 1° F. for every 300 to 350 feet, and is less the dryer the air. Alterations in temperature are less near the sea and less in the southern hemisphere. The higher the elevation and the colder the air, the less moisture does it contain. About the factors of ozone and electricity in the air little definite is known.

Climates are classified by Weber into marine, low level inland, and high level inland. These all have great variations in quality, depending upon their temperature, moisture, etc. As a general rule, warm marine climates and sea voyages are best for neurasthenic invalids of the irritable type. On the other hand, in atonic and anæmic conditions high inland climates are better, at least for a time. Such climates should not be too dry or windy.

In organic degenerative diseases of the nervous system, marine climates and low levels are better.

Germany, the Riviera, the Bermudas, the West Indies, Florida, and other Southern States are favorite places for sending neurasthenic Americans. Camp life in the Adirondacks or other forests is also found most useful.

Electricity in Nervous Diseases.

Physical.—Electricity is assumed to be a material like a fluid, perhaps a condition of the ether itself. It is not a force any more than water is a force, but it produces force by its movements. Electrical phenomena are the result of the strain or stress put upon the electrical fluid. Physicists assume that the electrical fluid exists in two conditions, positive and negative, and we speak of positive electricity and negative electricity accordingly. Under ordinary conditions these fluids are united and in equilibrium; but by certain agencies, such as friction, heat, chemism, etc., they can be separated. We assume that the condition of electrical equilibrium is that of zero, and that the earth's electricity is at zero. Positive electricity is raised above, negative electricity pulled below, this zero point. Electrical phenomena result from attempts of the fluids to become equalized or stable again at the zero point, just as the phenomena of heat result from differences of temperature and those of gravity from differences of pressure. The distance to which the electrical fluids are separated from the zero point is spoken of as the difference in "potential." This *potential* corresponds to the term "degrees" in measuring heat. Now, the greater the difference in potential, the greater the effort of the fluids to return to zero. High and low potentials correspond to high or low intensity of heat. *Tension* is the result of the widely separated fluids striving

to return to the zero point. It is the same thing as *difference of potential*, and the term may as well be dropped. When the two fluids, at different potentials, attempt to become equalized, they pass along certain paths and form *electrical currents*. Electricity will pass along any substance, but some substances conduct it more easily than others, and these are known as *conductors*. The relative value of different conductors is shown in the following table:

1. The metals; 2, charcoal, 3, plumbago; 4, dilute acids; 5, saline solutions; 6, pure water; 7, living animals; 8, flame. Of the metals, silver and copper are the best conductors. The human body would have about the same conductibility as the saline solutions, if it were not for the skin, which is a very poor conductor, especially when it is dry. Those substances along which electricity passes with great difficulty are known as non-conductors or *insulators*. The following is a list of some of these, the substances arranged in accordance with their relative value:

1. Caoutchouc. 2. Silk. 3. Glass. 4. Wax. 5. Sulphur. 6. Resins. 7. Shellac. 8. Dry air.

The electrical fluids may be kept by insulators at different potentials, the insulators preventing them from becoming equalized or reduced to the same potential. Electricity in this condition is called static. Its study is called electrostatics.

The electric fluid in motion is called dynamical electricity, and its study is electrodynamics.

Technical Terms.—There are certain technical terms which it is necessary to understand. *Electromotive force* (symbol, EMF) is the force which tends to set electricity in motion. An electric current results. The *current strength* (symbol, C) is the term used to express the capacity of the separated fluids to overcome resistance in their attempts to reach equilibrium or equalization again. This current-strength or simply the current, naturally, is in proportion to the strength of the electromotive force, which is constantly disassociating the electrical fluids and generating the current. If, however, as is always the case, the electrical fluid meets resistance in seeking equilibrium, the resistance diminishes its current. Hence we have the formula known as Ohm's law:

$$\text{Current strength} = \frac{\text{Electromotive force}}{\text{Resistance}}; \text{ or } C = \frac{EMF}{R}.$$

All bodies offer some resistance to electrical currents, and it is important to have some standard unit of resistance for the sake of comparison. Such standard unit has been adopted and is called an *ohm*. It is the resistance offered to a current by a certain piece of wire of definite size and length.

A *volt* is the unit of electromotive force, *i.e.*, it represents the force which will generate a certain amount of electricity in a second of time. A Daniell cell is of not quite one volt strength.

An *ampere* is the unit of working power or current strength. It is the current strength produced by one volt of electromotive force working against one ohm of resistance. A milliampere is one-thousandth of an ampere. A *watt* is the unit of work.

Density.—When a given current flows along from a large into a small conductor, the quantity in this latter conductor in a given section is greater and the current is said to be denser. The instrument by which the strength of a current is measured is known as the amperemeter: in medical practice, only fractions of the ampere are used, and the instrument is called the *milliamperemeter*. A *rheostat* is an instrument for interposing resistance in a current.

Electrical Appliances.—The batteries used in neurological practice are of three kinds: the static, the faradic, and the galvanic.

The static electrical batteries are mostly modifications of the Holtz influence machine. They are inclosed in glass to prevent the effects of moisture. The instruments made in this country for medical purposes will usually furnish electricity all the year round. A cheaper instrument, made on a different principle by Glaser, of Vienna, is recommended by Lewandowski. The battery accessories consist of an insulated stool and brass-ball electrodes with glass handles. The patient is placed on the stool, which is connected by a rod with one of the prime conductors. The battery being started, the patient becomes enveloped, as it were, with a layer of electricity which is at a very high potential and constantly flying off, being retained only by the dry, non-conducting air. The electrode held by the operator, and connected directly or indirectly to the other prime conductor, is now brought near the patient. The electrical fluid bounds to the zero point with such force that some of the metallic parts of the electrode are carried off and ignited, causing the disruptive spark. The patient thus is discharged of the fluid; but it continually reaccumulates upon him, and thus one can keep on drawing sparks from all parts of the body. The electric spark causes a muscular contraction and, a little later, a small punctate red spot. There is some pain connected with it, but as the electricity penetrates the body for only an infinitesimal period of time, it directly affects the viscera but little, and is not dangerous. Various ingenious electrodes have been devised for modifying the character of the static discharge, but they all have much the same effect as the spark.

By approximating the prime conductors so that they almost touch, and then connecting the outer surface of the two Leyden

jars which hang from the prime conductors with sponge electrodes, one gets the static induced current described by Dr. William J. Morton. The special peculiarity of this current, as well as of the spark discharge, is that it is made up of electricity at a very high potential, and that, being composed of oscillating or alternating currents of extremely short duration, the quantity of electricity is very small. The result is that when a muscle degenerates, it loses its irritability to the static current very early. The static current will thus reveal a beginning degeneration of nerve sooner than the other currents; it also promotes tissue changes more than other forms. It sometimes stimulates a greatly degenerated muscle, so that later the other forms will produce a contraction. It has a powerful psychical effect, and lends itself readily to quackery.

In *faradic medical batteries* the electricity is produced by induction. The chief elements are a cell and two coils of insulated copper wire. One coil is placed around the other, the outer coil being longer and of finer wire. The cell generates a current which in turn "induces" the electricity which is received by the patient. The original current is so arranged that it is being constantly broken and closed or "made" again. At both break and make, a current is induced in the inner coil of coiled wire; this forms what is called the *primary induced current* and is made up of a succession of short currents. This current, though theoretically alternating, is really a current of one direction. For the "make" current is opposed and nullified by the original or battery current. The same is true of the *secondary induced current* which is excited in the outer coil by the currents of the inner coil. Both currents are really composed of a rapid series of single currents going the same way, and they each have a positive and a negative pole. The primary current is one of lower tension, and is rather less strong. It can be used when the resistance of the parts is not great and a very powerful current is not needed. The secondary current is one of higher tension, it overcomes resistance better, and can be employed in connection with the wire brush and in anæsthesic conditions. It is also the current used in measuring the strength of the faradic application, as will be shown later. The current of the secondary coil resembles that of the static induced current. It has, however, a lower potential, vastly slower alternations, and more quantity. It can contract muscle, which the static current cannot affect. The secondary coil should be made of wire of a standard size, length, and layers of coil. A standard coil of wire 800 metres long, .225 mm. in diameter, wound on a spool 100 mm. long, is quite generally adopted now.

Faradic batteries are made with the zinc-carbon or Grenet cell, the Leclanché, the silver-chloride cell, or some modification of

these. For general use, the zinc-carbon cell is the most trustworthy; but the dry silver-chloride cell is the most convenient. A dry cell made with muriate of ammonia has of late come into use.

Galvanic Batteries.—There are two kinds of these in practical use. The one includes the cells which act as soon as the circuit is closed; the other includes those which act only when the elements composing the battery (*e.g.*, zinc, carbon, platinum, copper, etc.) are dropped in the exciting fluid. The former class (known as two-fluid cells) are not touched except to renew water or add some chemical. In the latter (the single-fluid cells), one of the elements is always taken from the fluid when the current is not in use. The first class of cells has a much weaker chemical

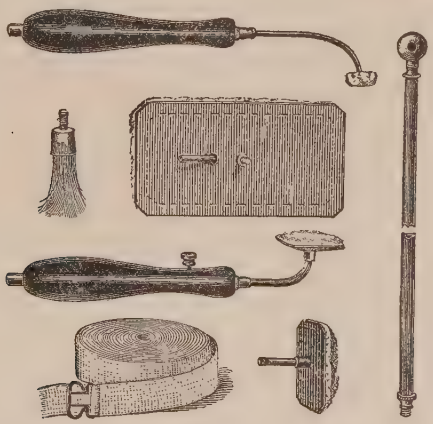


Fig. 30.—Author's Electrode Set (H. E. Stammers, New York).

action and evolves less electricity in a given time. It includes the Daniel cell, the gravity cell, the Leclanché cell, and the silver-chloride cell. Among the second class or single-fluid cells, the zinc-carbon cell, known as the Grenet or Stohrer's cell, already referred to, is most used. The best portable batteries are made of the zinc-carbon cell or the dry chloride of silver cells. Stationary or office batteries are best made with the Leclanché cell or some modification of it; or the dry silver-chloride cell may be used. The electric-light current can be utilized to supply continuous and interrupted currents and for purposes of the cautery. It is expensive.

The ordinary accessories to the faradic and galvanic batteries are electrodes, rheostat, and milliamperemeter.

The electrodes needed for ordinary purposes are:

1. An indifferent electrode measuring 5 cm. by 10 cm. 2. A normal electrode, 10 sq. cm. 3. A unit electrode, 1 sq. cm. 4. A

soft wire brush. 5. Three handles: one 10 cm. and one 40 cm. long, one short handle with an interrupter. 6. A milliamperemeter. 7. A rheostat. (See Fig. 30.)

Methods of Application.—Static electricity is applied for fifteen or twenty minutes daily or tri-weekly. For general tonic or sedative effects, sparks are drawn from all parts of the body except the face. In paralysis or spasm or pain, sparks are applied to the affected area. For headaches and cerebral paræsthesiæ, the electrical breeze is very useful, but it must be strong.

The faradic and galvanic currents are used for about the same time and intervals as the static. In some cases, however, the galvanic current should be given daily or even two or three times a day. As a rule, a course of electrical treatment should be continued for six to eight weeks, and then discontinued for a time.

The special methods used in applying these currents are:

1. General galvanization and faradization or general electrization.

2. Local electrization by galvanization of the brain, of the neck, of the spine, of the special senses, limbs, and viscera. Or by faradization of the neck, spine, limbs, and viscera.

3. The combined faradic and galvanic currents. These are given by means of the De Watteville switch. General and local electrization can be given in this way.

4. The polar method. This is employed chiefly in using the galvanic current. The indifferent electrode is placed on the sternum or back, and the other electrode applied wherever indicated.

5. Cataphoric electrization by means of Peterson's electrode.

6. Electrolytic applications are used in enlarging strictures and affecting inflammatory deposits and neoplasms.

In general electrization, whether galvanic or faradic, the indifferent electrode is placed on the sternum, feet or back, and the other pole is carried over the limbs, trunk, neck, and, if indicated, the head. In some cases, however, the two electrodes are applied together upon the different muscles of the body. In local electrization, the large electrode may be applied on an indifferent spot, and the other applied to the affected limb or limbs, or the two electrodes may be used together on the same segment of muscles. The details for galvanizing the brain, special senses, and viscera must be obtained from special text-books.

When an electrode is held steadily upon a part, it is called *stabile;* when it is moved over it, it is called *labile.* The positive pole is called the anode (An), the negative the cathode (Ca). The size of an electrode is indicated in square centimetres.

A formula for applying electricity may be written thus:

Anod. galvanization, 10 ma. 5–10 cm. daily, stabile.

This means that the positive pole of the galvanic current is to be applied steadily at a given place daily for five minutes with an electrode of ten square centimetres.

The terms "ascending" and "descending currents" are rarely

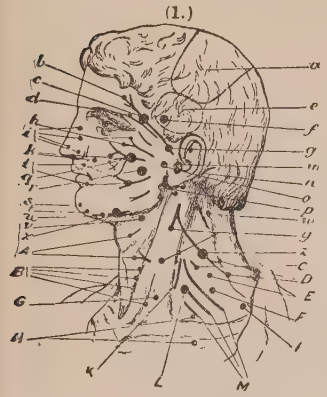


FIG. 31.

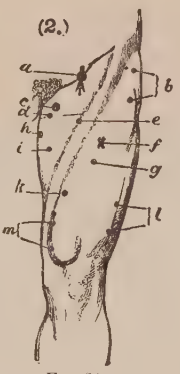


FIG. 32.

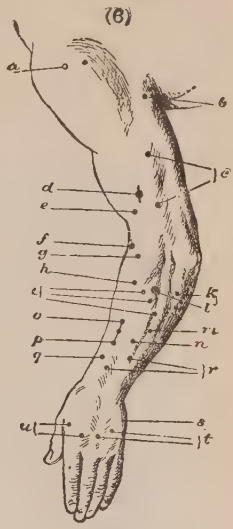


FIG. 36.

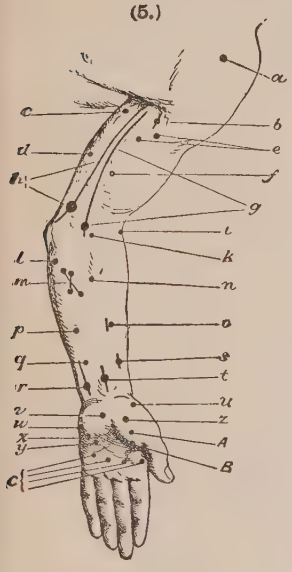


FIG. 35.

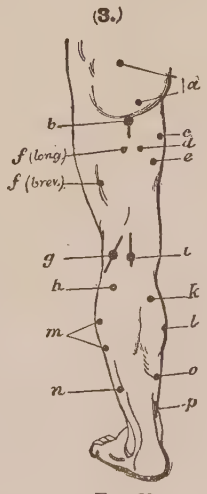


FIG. 33.

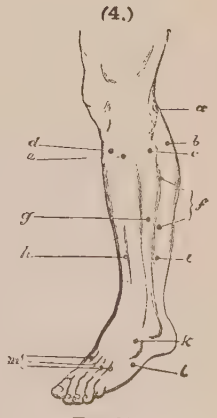


FIG. 34.

FIGS. 31 TO 36.—CUTS SHOWING MOTOR POINTS. The letters refer to points electrization of which stimulates certain muscles to contract. These points vary much in different persons. For details see De Watteville's "Medical Electricity."

used, the name of the pole being employed instead. Thus, anodal galvanization of the brain or arm means that the positive pole is applied at these localities. With the faradic and static currents, neither the pole nor the direction of the current makes much difference.

Electro-Diagnosis.—When a motor nerve is cut off from its centre in the spinal cord, or when this centre itself is diseased, the nerve and later the muscle undergo a degeneration. As a result of this, their reaction to electrical currents is changed, and we get what is termed "partial degeneration reactions" and "complete degeneration reactions" according to the degree of disturbance. These reactions are due mainly, if not wholly, to the degeneration in the terminal nerve fibres and motor end plates in the muscle. When the muscle alone is diseased, the reaction is not changed until very late. The change in irritability is due to the fact that as the nerve fibre wastes it takes an electric current of comparatively long duration and considerable strength to stimulate it.

The first effect is to lose its contractility or reaction to weak currents, then to extremely rapid, short currents like the static, then to the faradic, and last to the galvanic. Such change is known as the *quantitative alteration* in electric irritability.

But besides this, the nerve and muscle are affected in a different way by the different poles of the galvanic battery. In normal nerve and muscle, a contraction is caused more readily by the negative pole than by the positive. But muscles with degenerated nerve supply sometimes respond as well or better to the positive pole. This forms what is called the *qualitative* or *serial change* in the irritability of the muscle.

Finally, degenerated muscles respond more sluggishly than normal to the galvanic and faradic currents. The contraction, instead of being sharp and jerky, is sluggish and almost tetanic. This is called the *modal change* in irritability.

The *qualitative* change is only gotten by placing the active electrode over the muscle, but the *quantitative* and *modal* changes may be gotten by placing the electrode over the nerve as well as over the muscle. In describing these changes, the following abbreviations are used:

DeR = degenerative reaction.

AnCC = anode or positive-pole closure contraction.

CaCC = cathode or negative-pole closure contraction.

AnOC = anode opening contraction.

CaOC = cathode opening contraction.

Te = tetanus.

D = circuit is closed and current flowing.

ADTe = tetanic contraction while the positive pole is applied and the circuit closed.

The sign > means greater than; <, less than. Thus AnCC > CaCC means anode closure contraction is greater than cathode closure contraction.

Degenerations in nerve do not occur except in lesions of the nerve or spinal cord, and in very late stages of primary atrophy of muscles. Hence when one finds degenerative reactions, he can exclude disease of the brain, functional disease, and primary disease of the muscle. The following rules may be formulated for testing for degenerative reactions:

Use the faradic current first.

The Faradic Current.—Use a secondary induction coil of .225 mm. diameter, 800 metres long. The distance over which the coil moves is divided into a hundred parts. The strength of current is indicated by percentage or millimetres. In many scales it takes 30 to 40 mm. of CD or coil distance to cause a muscular contraction. Record the minimum necessary for muscular contraction, using the same electrodes and in the same way as in testing with galvanism.

The Galvanic Current.—1. Place the indifferent pole over the sternum, and a 10 sq. cm. electrode over the muscle. 2. Pass the current for one minute. 3. Then find the minimum strength needed for a cathode closure contraction. 4. Then for an anode closure contraction. Repeat this test three times. 5. With a given current, note whether the cathode closure contraction is stronger than AnCC, or otherwise. Test this three times. 6. Note the character of the contraction, if sharp or sluggish. 7. Test nerve in same way.

The qualitative changes may be expressed by a formula like AnCC = or > CaCC, *i.e.*, the positive-pole closure contraction is equal to or greater than the negative-pole closure contraction. Or, better, the minimum strength of current required to cause a contraction in the muscle is recorded for the positive pole and for the negative. Thus:

AnCC 5 ma. or 8 cells.
CaCC 4 ma. or 6 cells.

Blanks for such records are used in the Post-Graduate Clinic, like the following:

RECORD OF ELECTRICAL REACTIONS.

Case of..... *No.*.....

MUSCLE. NERVE.	REACTION TO GALVANIC CURRENT. { ELECTRODE ....SQ. CM.		REACTION TO FARADIC CURRENT. 2d ind. coil.		DATE.
	Right Side.	Left Side.	Right Side.	Left	
Ant. tibial muscle.	AnCC 8 ma. CaCC 6 ma.	AnCC 5 ma. CaCC 7 ma.	0	10 CD	June 6, 1892.

The following table modified from De Watteville and diagram (Fig. 37) show the diseases in which degeneration reactions may be expected.

TABLE SHOWING THE LESION, ITS RESULT, THE NAMES OF THE DISEASES, AND THE ELECTRICAL REACTIONS.

Lesion of—	Result.	Disease.	Electrical Reaction as to Qualitative.
1 to 2½. Cortex to cord.	Paralysis, contractures.	Hemiplegia from hæmorrhage. Embolism, tumors, lateral sclerosis.	Nerve: normal. Muscle: normal.
3, 4, and 5. Cornua.	Paralysis, degenerative atrophy of nerve and muscle.	Acute and chronic anterior poliomyelitis.	Nerve: DeR. Muscle: DeR.
2 to 2½. 3 to 5. Lat. cols. and ant. corn.	Paralysis, contractures. Degenerative atrophy of muscle.	Amyotrophic lateral sclerosis.	Nerve: normal } DeR. Muscle: partial }
5 to 6. Trophic cord centres.	Degenerative atrophy of muscle. Paralysis from wasting of muscle. Later, degeneration of nerve.	Progressive muscular atrophy (spinal form), bulbar paralysis; (?).	Nerve: normal. Muscle: or partial DeR. When disease is advanced, in early stages, etc.
Nerve.....	Paralysis: degenerative atrophy of nerve and muscle.	Neuritis; from wounds, toxæmia or pressure.	Muscle: DeR. Nerve: DeR.
Muscle..... 6.	Wasting, paresis.	Simple atrophy; primary or idiopathic myositis, Juvenile form of progressive muscular atrophy; pseudo-muscular hypertrophy; other types of primary myopathies.	Nerve and muscle normal until late in the disease.
Nerve and muscle.	Paresis and atrophy.	Rheumatic atrophy and paresis.	No DeR. unless severe.

It should be said, finally, that it is the sluggish contraction which is the most important element in showing degeneration; also that it is the muscle which should be tested most carefully, as only over it does one get the qualitative changes.

Therapeutics.—Electricity is used as a counter-irritant and as a general mechanical tonic in states of muscular and nervous weakness. It is used in paralysis, spasm, and pain, and for its supposed specific action in certain functional and organic diseases.

The faradic and static currents have a counter-irritating, stimulating, and excito-reflex effect. The galvanic current has a sedative and anti-spasmodic effect.

Electrolytic, cauterizing, and cataphoric effects are also pro-

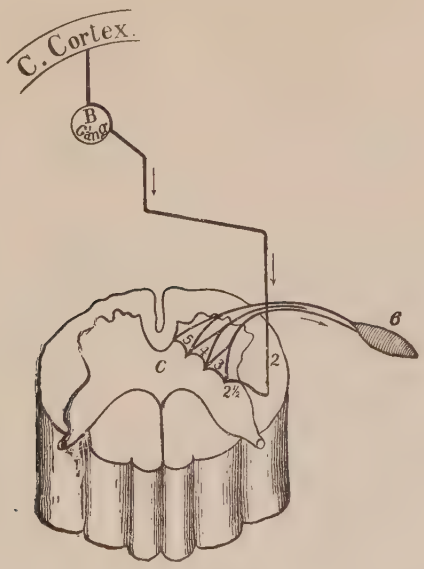


Fig. 37.

duced, but are rarely needed by the neurologist. A considerable portion of the effects of electricity are psychical, but they are not the less real or valuable.

CHAPTER VI.

GENERAL DISEASES OF THE PERIPHERAL NERVOUS SYSTEM.

THE disorders which affect the peripheral nerves are hyperæmia and anæmia, degeneration, inflammation, functional disorders, new growths.

I shall describe here those disorders of which general facts can be stated, no matter where the trouble is located. Such disorders are hyperæmia and anæmia of nerves, degenerative processes, inflammation.

Next will come a description of diseases which affect the whole or a large part of the whole peripheral nerves simultaneously. Such disorders are multiple neuritis, neuralgia, and certain other neuroses.

HYPERÆMIA.—*Etiology.*—This is a condition rarely recognized clinically. The causes are cold, injury, neighboring inflammation, rheumatic, gouty poisons and paralytic vasomotor neuroses, metallic and other poisons.

Symptoms.—The symptoms are those of burning, numbness, and other paræsthesiæ; tenderness, pain, and muscular weakness. Doubtless many forms of neuralgia are associated with hyperæmia.

Pathological Anatomy.—The nerve in hyperæmia is reddened, injected, and may be the seat of minute hemorrhages.

Treatment.—As hyperæmia and anæmia are always secondary conditions, the treatment is mainly based on the cause. The ice-bag, leeches, counter-irritation, cupping, and massage may be resorted to in hyperæmia; local stimulants, hydrotherapy, massage, nitroglycerin, iron, and iodide of potassium may be given.

ANÆMIA OF NERVES.—*Etiology.*—This occurs in conditions of general anæmia and cachexia, in spasmodic vasomotor neuroses, in obstruction of blood-vessels from any cause, and in obliterating arteritis.

Symptoms.—Often no especial symptoms result. Here again, however, we may trace the cause of pains and neuralgias, especially those of the later periods of life when arteritis is present.

Degeneration of Nerves.

This is a process in which the nerve fibres gradually die; the myelin sheath and axis cylinder disappear, leaving only a strand of connective tissue.

Etiology.—It is caused by destruction of the trophic centres of the nerve or by cutting the nerve off from it; or by direct action of toxic and inflammatory and destructive agents on the nerve fibre. Diseases of the spinal cord, injuries of nerves, and toxic agents are the special causes. There is occasionally also a primary atrophy of peripheral parts of nerves in wasted and disused muscles.

The symptoms of nerve degeneration are those of sudden or gradual enfeeblement and loss of the nerve's function. There may be some evidence of irritation, such as pain and spasm. Paralysis, anæsthesia, and various trophic and vasomotor disturbances occur in the part affected.

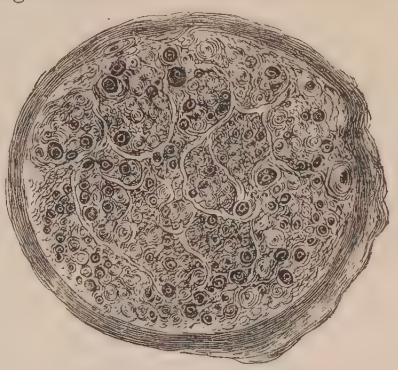


Fig. 38.—Simple Atrophy of Nerve in Marasmus.

Degeneration of nerves is usually secondary to or associated with other diseases, and its special symptoms will be described elsewhere.

Pathology. — There are three forms of nerve degeneration: 1. Primary; 2. Secondary; 3. Neuritic or toxic.

1. The primary form is rare, slight in extent, and of little clinical significance. In it there is simply a gradual wasting and disappearance of the axis cylinder and myelin sheath. It occurs in wasting diseases and as part of locomotor ataxia (Fig. 38).

2. Secondary degeneration or Wallerian degeneration. This form occurs when the nerve is cut across, or compressed, or destroyed by inflammation, neoplasms, or injuries. The peripheral end of the cut nerve shows a loss of nearly but not quite all the fibres as far as its termination. In the central end, the degeneration only ascends to the first or second node of Ranvier. A few fibres, however, degenerate as far as the spinal cord. These are afferent in function, they have their trophic centre in the periphery, and correspond to the undegenerated fibres in the peripheral part of the cut nerve.

When the section is made between the spinal ganglia and the cord, the fibres all degenerate toward the cord, and even enter it, but the peripheral fibres do not degenerate. Hence the spinal ganglia are the trophic centres of most of the sensory nerves.

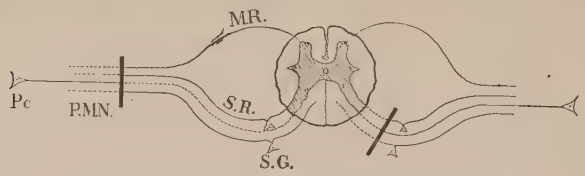


Fig. 39.—Cut showing the Effects of Section of Mixed Nerves and Sensory Root. P.M.N., Mixed nerve; S.G., spinal ganglion; M.R., motor root; S.R., sensory root; *P.c.*, peripheral trophic cell. The dotted line shows the degenerated nerves.

A few, however, have their trophic centre in peripheral nerve cells.

Degeneration occurs in the motor nerves, also, when the cells of the anterior horns are destroyed. Hence these cells are the trophic centres for all motor nerves.

The essential part of the nerve fibre, the axis cylinder, is simply a prolongation of the process of a nerve cell. Its next essen-

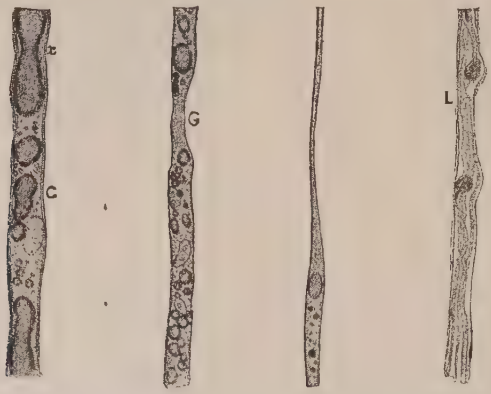


Fig. 40.—Showing Different Stages in the Process of Nerve Degeneration on Second, Third, Sixth, and Ninetieth Days after Section (Ranvier).

tial part is the myelin sheath. This is of epiblastic origin and consists of a hollow cylinder inclosed in a thin membrane and containing a fatty substance.

In degenerative processes of peripheral nerves the medullary sheath is first affected, then the axis cylinder, least and last the neurilemma. The myelin becomes turbid, splits up into frag-

ments and droplets. The axis cylinder also breaks up into fragments or swells up and becomes liquefied. Extravasated leucocytes pick up the products of disintegration and form fat-granule cells. The neurilemma and its nuclei usually remain intact. The nerve during this time shrinks in volume and looks grayish and translucent, or grayish-red. The nerve finally becomes only a fibrous cord. Changes can be seen in the nerve within forty-eight hours, and by this time its irritability, which was first slightly increased, is lost. In about two weeks the disintegration of the myelin sheath and axis is practically complete.

The process of degeneration takes place at about the same time throughout the whole length of the nerve. The motor end plates in the muscles are affected a little the earliest. The fibres of the central stump are affected as far as the first or second nodes of Ranvier. Later, the central stump may degenerate somewhat from disuse. About the cut end, however, little bulbous tumors may develop, which contain numerous nerve fibrils and connective tissue. The general law is that nerves degenerate in the direction in which they carry impulses, but this is not always the case, as is seen with peripheral sensory nerves.

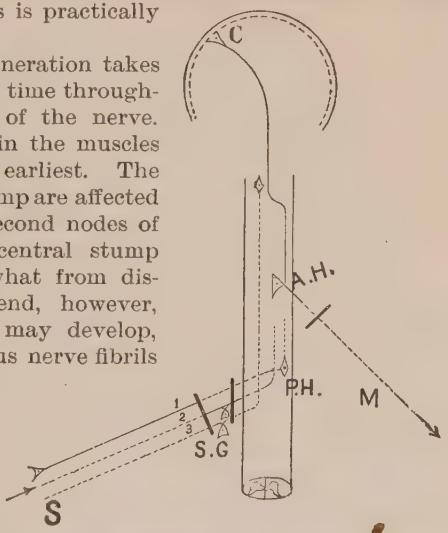


Fig. 41.—Showing the Relations of the Nerves to their Trophic Centres. S., Sensory nerve; M., motor nerve; S.G., spinal ganglion; P.H., posterior horn; A.H., anterior horn; C., cerebral cortex. The dotted line shows the degenerated nerve.

The relations of nerve fibres to their centres and the effects of section is shown in the accompanying cuts.

Summary: Peripheral nerve fibres degenerate when cut off from their trophic cells.

The degeneration begins at once throughout the length of the nerve. Loss of function occurs in forty-eight hours. The degeneration is practically complete within two or three weeks.

The myelin sheath and its nuclei are affected first, the axis cylinder next.

The degeneration takes place in the direction in which the nerve impulse runs except in peripheral afferent nerves.

The final stage is one of nerve atrophy.

Within *the central nervous system* the degeneration always occurs in the direction of the nerve impulse. The axis cylinder is first affected. There is sometimes a preliminary swelling or hypertrophy of the axis cylinder. Degeneration with calcification of the nerve fibres sometimes occurs. In associative or commissural fibres the degeneration only extends part of the length of the nerve.

3. Neuritic or toxic nerve degeneration. This form occurs in connection with neuritis, and will be described under that head.

Its chief characteristics are that the degeneration attacks the nerve in segments, that the axis cylinders are not so much affected, and the myelin breaks up into small fatty droplets instead of into large masses.

Degenerative processes in the non-medullated nervous fibres have been observed in the fine fibres of the cornea and in the submucous and myenteric plexuses of the alimentary tract.

Regeneration of nerves is a process that usually follows degeneration. It occurs only in peripheral nerves—very little, if at all, in the nerves of the central nervous system of mammals. It is a unique process, in that the nerve is the only specialized tissue that can grow again after being destroyed. Regeneration occurs whenever the trophic centres are healthy, when the mechanical obstacles to a union of the divided fibre are not too great, and when the peripheral nerve is not too completely atrophied. It occurs most quickly, therefore, when the cut ends are carefully apposed and when the separation has not lasted for a long time, *i.e.*, for years. It progresses always from the *central* end toward the periphery. The fibres of the central stump grow out into the degenerated peripheral fibre. Union by *first intention* or *second intention* never occurs. Human nerves cannot be made to unite physiologically, but only anatomically.

Regeneration may be complete in a few months in short nerves. In the sciatic it may take one or two years. When regeneration takes place, the axis cylinders of the central stump swell and divide into a number of new cylinders which pierce or creep around the intervening tissue, enter in bundles the peripheral nerve, and become inclosed in new myelin sheaths and neurilemma.

INFLAMMATION OF NERVES—NEURITIS.

Anatomically and clinically, there are two forms of neuritis:

1. Neuritis of single nerves or groups of nerves.
2. Multiple neuritis.

Pathologically, there are two forms of neuritis:

1. Interstitial neuritis and perineuritis.
2. Diffuse neuritis with parenchymatous degeneration.

Etiologically, there are two forms:

1. Toxic neuritis, from alcohol, arsenic, lead, etc.

2. Infective neuritis from pyogenic organisms, infective fevers, syphilis, tuberculosis, leprosy, etc.

I shall follow the anatomical and clinical classification and describe, first, neuritis of single nerves or groups of nerves; next, multiple neuritis.

Neuritis of Single Nerves or Nerve Groups.—*Etiology.*—The most frequent cause of neuritis by far is injury, such

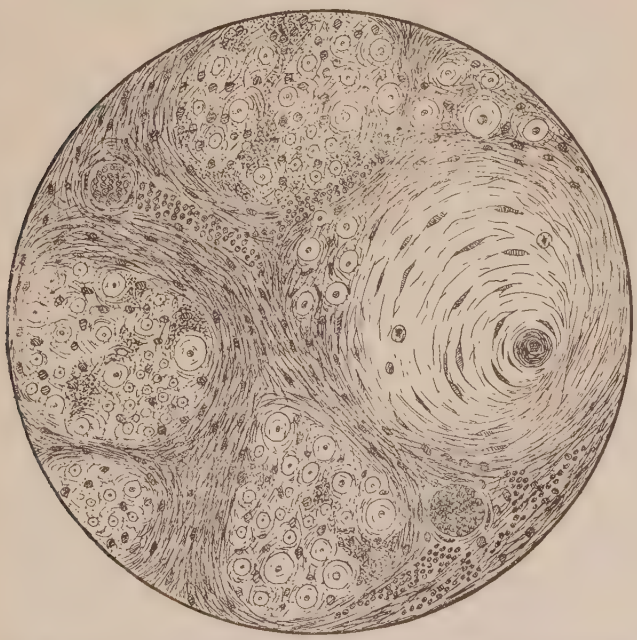


Fig. 42.—Acute Infectious Neuritis, showing hæmorrhage, connective-tissue proliferation, diseased nerve fibres, and obliterated vessel (Rosenheim).

as compression, tearing, stretching, cutting, etc. Next comes, perhaps, lead-poisoning; after this, various infections, such as typhoid fever, variola, diphtheria. Tuberculosis, leprosy, syphilis, are also causes.

Symptoms.—These are such as result from irritation and paralysis of the nerves affected, and will be described under the head of Special Forms.

Pathology.—Injury may not cause a true neuritis, but only a secondary degeneration. When inflammation does occur it is of

an interstitial or perineuritic type, and it may be acute or chronic. In the former case there is hyperæmia, with sometimes extravasation of blood. An exudation occurs into the fibrous framework of the nerve, with migration of leucocytes. The inflammation may become suppurative or gangrenous. If severe, it destroys the nerve fibres; but oftenest the axis cylinders are not destroyed, and recovery takes place. Chronic interstitial neuritis and perineuritis are accompanied with hyperplasia of the connective tissue, compression and more or less destruction of the nerve. It may ascend or descend, and it is called, accordingly, *ascending*, *descending*, or *migrating* neuritis. It may affect only certain segments of the nerve, when it is called *segmental* neuritis or disseminated neuritis. Tubercular and syphilitic neuritis are of the chronic interstitial or diffuse type. These latter forms rarely involve peripheral nerves, but rather the intra-cranial parts of the cranial nerves and the spinal nerve roots in meningeal tuberculosis or syphilis. A syphilitic peripheral multiple neuritis is, however, thought to occur sometimes. Leprous neuritis is a very typical form of proliferating chronic perineuritis. Cancerous neuritis sometimes occurs, and it is of the diffuse type, though sometimes an actual cancerous process invades the nerve.

The course, duration, prognosis, diagnosis, and treatment of the above types of neuritis will be given under the special heads.

Multiple Neuritis (Polyneuritis, Pseudo-Tabes, Alcoholic Paralysis Beri-beri, Ignipedites, Acrodynia).

This is an inflammatory and degenerative affection attacking a number of nerves simultaneously and characterized chiefly by wasting and painful paralysis or by ataxia, with some motor weakness. It generally runs a subacute course.

Forms.—Multiple neuritis may be of the paralytic or motor and of the ataxic or sensory type. There are also endemic types and acute pernicious forms.

Etiology.—The disease is due to infections, to auto-toxic agents, and to chemical poisons.

The special infections are those causing the disease beri-beri, the infection of diphtheria, and probably almost all of the exanthematous fevers, as well as of puerperal and other septic fevers, cholera, ankylostoma, cerebro-spinal meningitis, and possibly phthisis and syphilis. The most common infection in this country is diphtheria. In the West Indies malaria, in France an infection causing acrodynia, in Japan, China, and various other tropical regions the infection of beri-beri are causes. Probably an independent specific infection of unknown character sometimes is a cause.

The autochthonous or auto-toxic poisons are those of rheumatism and diabetes. An incomplete form of polyneuritis is sometimes seen as the result of exposure to cold and wet, as in handling ice. Conditions of inanition sometimes seem to lead to it.

The toxic causes from without are those of alcohol, arsenic, lead, anilin, ergot, bisulphide of carbon, illuminating gas, mercury, and phosphorus. Practically, alcohol causes multiple neuritis in the great majority of cases (20 out of 31). In this country rheumatism and the infective fevers, especially diphtheria, are not infrequent causes, and the metallic poisons, with arsenic leading, come last.

Sex.—Multiple neuritis occurs oftener in the female than in the male. This is due to the fact that alcoholic neuritis, which is the common form, affects women oftener. Beri-beri occurs much oftener in the male.

Age.—Multiple neuritis is essentially a disease of early adult life. Almost all cases occur between adolescence and the period of degenerative changes, twenty to forty-five. Young children are very rarely subject to it. The youngest age reported is fourteen years (Suckling). A few cases have occurred in persons over sixty; but they are but little less susceptible than children. In beri-beri, three-quarters of the cases are between the ages of fifteen and twenty-five, 6% in children, 1.2% in old people (Harada).

Season and Climate.—The sporadic forms of polyneuritis from alcohol and various poisons and infections occur without much reference to a seasonal influence. Probably more cases occur in spring and fall, owing to sudden changes in temperature. Beri-beri occurs oftener in warm weather. Epidemic influences like that causing cerebro-spinal meningitis may increase the number of cases of multiple neuritis. Practically, in this country, the question of drink settles the question of the distribution of polyneuritis. It is rare in the temperate rural districts and smaller towns, and much rarer in native Americans than foreigners.

Sexual excesses, exposure to cold and wet, insufficient diet, are among the helping causes.

Symptoms and Course.—Polyneuritis usually begins somewhat acutely, but runs a slow course of several months. Very rarely it runs a sudden course, ending fatally. We have, therefore, the ordinarily *subacute multiple neuritis* and acute *pernicious multiple neuritis*. The endemic infectious forms show other types.

The following is the classification followed:

1. Subacute, multiple neuritis.
   - Motor type.
   - Sensory type.
   - Endemic forms: Beri-beri, etc.
   - Malarial forms.

2. Acute malignant multiple neuritis.

1. This is the type of neuritis produced by alcohol, infections, poisons, and rheumatic influences. It is usually chiefly a motor trouble, but sometimes takes the sensory or pseudo-tabetic form.

Prodromata.—The disease often begins with prodromata lasting several weeks. The patient suffers from numbness, slight pains, and weakness affecting especially the lower extremities. In other cases the attack comes on suddenly, and in a short time the most marked symptoms appear.

Fig. 43.—Alcoholic Paralysis, with Foot Drop and Wrist Drop.

Onset.—There may be a fever for three or four days, with a temperature of 103° or 104°. This is not the rule. Numbness and pains attack the feet and legs, rarely extending much above the knees. The muscles and nerves are very tender. The fingers, hands, and arms are often similarly but less affected. At the same time the skin becomes reddened or slightly œdematous. The muscles of the legs grow weak, and in a day or two the patient may be unable to stand. In a week or two there may be a complete loss of power in the anterior tibial muscles and a lesser degree of paralysis in the extensors of the hand. Nearly all of the leg and forearm muscles become eventually involved. Atrophy sets in at the same time and very severe pains are present. The motor cranial nerves are in rare cases affected, and paralysis of the facial or of the third, fourth, or sixth nerve has been seen. When the disease is fully developed, which is within a fortnight, there is paraplegia with foot drop, some degree of wrist drop, muscular atrophy, and slight œdema, especially of the feet. The skin-reflexes are often, the knee jerk and elbow jerk always lost. There is some tactile anæsthesia, often with

hyperalgesia. Temperature and pain sense are also lessened and slowed in transmission. The anæsthesia occurs in patches or diffusely. Muscle and articular sense are lost in the sensory or pseudo-tabetic form, and are usually somewhat involved in the ordinary paralytic form. Pain and sensitiveness continue.

The nerves lose their irritability and the muscles show degeneration reaction, partial or complete, the characteristic being a great variability of reaction over different groups of nerves and at different stages of the disease, and an early loss of faradic and lessening of galvanic irritability. There is sometimes retinal hyperæmia and even optic neuritis. Of the visceral nerves, the vagus seems oftenest to show signs of involvement, in rapid pulse and disturbances of respiration. The sphincters are rarely involved. In such cases there is, perhaps, involvement of the cord or of the abdominal and pelvic splanchnics. In alcoholic and occasionally in other forms of neuritis, mental symptoms, such as a low, muttering delirium, are very often present, and occasionally a well-marked confusional insanity develops.

The disease usually reaches its height in a week or two and then starts on a chronic course; but it sometimes happens that exacerbations occur, or that a paralysis and atrophy progress for several weeks before regression begins. In alcoholic cases there is often great general prostration; the patients lie for several weeks in a delirious condition, and finally develop pneumonia and die.

Some further details should be added.

Motor Symptoms.—The characteristic paralysis of multiple neuritis is a quadruplegia, all four extremities being involved. The special characteristic is the foot drop, which is indicative of alcoholic neuritis, just as wrist drop is of lead palsy. The paralysis is typically a peripheral one. It affects the feet and legs, hands and forearms. It usually involves the anterior tibial muscles more than the calf muscles, but sometimes the reverse occurs. The muscles become wasted and flabby. They soon lose in bad cases all reaction to faradism, and they require a strong galvanic current to produce a contraction. In anterior poliomyelitis, on the other hand, the diminution in galvanic irritability comes on only after weeks or months. Hence an early loss of galvanic as well as faradic reaction is an important sign of neuritis. As the nerve and muscle recuperate, the galvanic irritability increases. After a time, if the paralysis is great, contractures occur. The feet are extended, the legs are flexed on the thighs, and are almost immovable, and the patient's condition is most pitiable.

Sensory Symptoms.—Numbness, hyperæsthesia, severe pains (dull and sharp), burning sensations, great tenderness, all occur, and are very marked symptoms. They are felt mostly in the feet,

legs, and hands. Hyperæsthesia is usually followed by anæsthesia to touch and somewhat to pain and temperature. The transmission of these latter two sensations is delayed. The anæsthesia sometimes occurs in patches, at other times diffusely over foot, leg, and hand. Muscular and articular anæsthesia are common, and in the sensory form are the dominant symptom, causing an ataxia of gait and station. The other special senses are not affected except in rare cases, in which there is optic neuritis.

Vasomotor and Trophic Symptoms.—There is often œdema, sometimes redness of the skin; occasionally the epidermis of the soles and palms peels off. Glossy skin and profuse perspiration are rare. Eruptions and ulcers do not occur.

Mental Symptoms.—The most common mental disturbance is that so often seen in acute alcoholism, viz., a muttering delirium. This is associated with great general vital depression.

If a true insanity develops, it also resembles, as a rule, alcoholic insanity or acute confusional insanity. The characteristic symptoms are a curious degree of forgetfulness, together with many and varying delusions rapidly succeeding each other. These often relate to the pains and paræsthesia from which they suffer. They think that there are gloves on their hands or that something is on their feet. They often think that they have been out walking or riding. They are talkative, incoherent, and sleepless.

Organic Centres.—The bladder is ocasionally affected for a short time, the other centres not at all. This freedom from involvement of the sphincters is an important characteristic of the disease in distinguishing it from myelitis. From the foregoing it will be seen that the dominant symptoms are paræsthesia, pains (burning, lancinating, and dull), muscular tenderness, some anæsthesia, paralysis affecting especially the lower extremities and causing *foot drop*, muscular wasting, with degeneration reactions; with no involvement of the sphincters; sometimes peculiar mental disturbances.

The sensory or pseudo-tabetic type of multiple neuritis is caused less often by alcohol and more often relatively by the metallic and infectious poisons. The general course is much like that of the paralytic form, but there is less paralysis, and on the other hand there are more of the burning, tearing pains, a greater degree of anæsthesia, with a very decided muscular anæsthesia causing symptoms of a subacute locomotor ataxia. The paresis, muscular wasting, trophic changes, such as shedding of the epidermis and electrical reactions, serve to distinguish the disease. A double facial paralysis sometimes complicates this type.

Endemic and Epidemic Types (*Beri-beri or Kakke, Ignipedites, Acrodynia, Malarial Multiple Neuritis*).—Beri-beri or

endemic multiple neuritis is seen in this country rarely, and only by accident. Beri-beri is the Indian name; kakke, meaning "the leg disease," is its Japanese name. Ignipedites is a name given by Indian physicians to probably the same disease. French physicians gave the name of "acrodynia" to an epidemic disease which prevailed in France and the Crimea in the early part of this century. It was probably multiple neuritis. There are various types of this disease, in some of which the neuritic symptoms seem subordinate to those of other organs. The forms described by Scheube and Taylor are:

The acute pernicious, the acute or subacute benign, the atrophic or dry, and the dropsical or wet.

The symptoms generally resemble those of multiple neuritis, as already described, plus œdema, extensive serous effusions, and gastro-intestinal disorders. The paralysis affects especially the lower extremities, but in beri-beri there seems to be an especial tendency also to involvement of vasomotor and visceral nerves.

Malarial Multiple Neuritis.—Jamaica seems to be the only place in which the malarial poison produces an endemic neuritic paralysis (Strachan), and the causation in these cases is not yet demonstrated. Dr. Strachan's description of the symptoms of what he terms malarial peripheral neuritis shows it to be quite extensive, often involving trunk and cranial nerves, and accompanied with much pain and wasting. Cramps and skin eruptions are often noted, complications that do not occur in the ordinary types.

2. Acute Pernicious Multiple Neuritis.—There is a form of multiple neuritis which comes on suddenly, progresses rapidly, and causes death in a few days or weeks. These cases usually show the ordinary symptoms of neuritic paralysis, with final involvement of the cardiac and respiratory nerves, causing death. The agent in these cases is apparently of the nature of sepsis. The neuritis is interstitial and hemorrhagic. Other cases of acute pernicious multiple neuritis take the form of acute ascending or Landry's paralysis. Here there are few sensory symptoms, no electrical changes or atrophy. The disease is due to an infectious poison which overwhelms the system before it has time to set up any inflammation or organic change.

Pathology.—In multiple neuritis the disease affects the periphery of the nerves most, and extends up, very rarely reaching the roots. The anterior tibial and musculo-spiral nerves on the two sides are oftenest and most diseased.

The process when mild in grade resembles a secondary degeneration following section of the nerve. In severer cases there is evidence of interstitial inflammation as well as degeneration.

This process, however, varies in degree at different points of the nerve's course. Hence it has been called segmental or disseminated neuritis. In some of these cases and in all acute pernicious cases there is still more interstitial inflammatory change; small hemorrhages occur, exudation takes place, and collections of leucocytes about the vessel walls and among the nerve fibres are seen. The muscles supplied by the diseased nerves undergo atrophy. This is usually simple and non-inflammatory. But

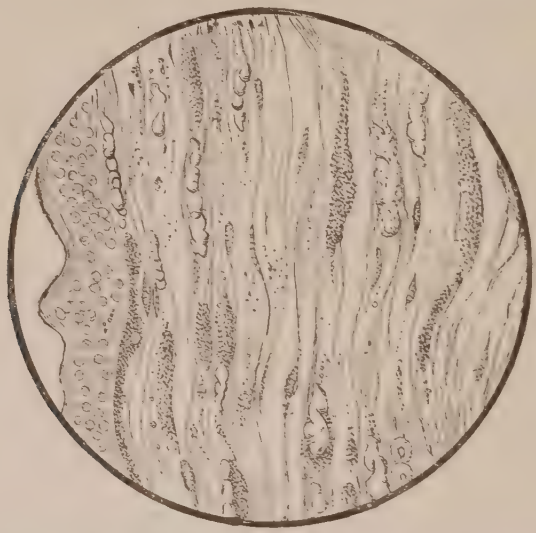


FIG. 44.—LONGITUDINAL SECTION OF A NERVE IN MULTIPLE NEURITIS, showing rich proliferation of nuclei (Leyden). The process here is inflammatory as well as degenerative.

sometimes there is an interstitial myositis with exudation compressing the fibres (Senator).

The spinal cord is, as a rule, healthy, or presents minor and secondary changes.

It will be seen, therefore, that in multiple neuritis there may be: (1) Simple degeneration; (2) degeneration with some evidences of interstitial neuritis (degenerative neuritis); (3) decided interstitial neuritis with degeneration of nerve fibres.

The changes occurring in nerve degeneration have been described. In the second class of cases—degenerative or, as it is sometimes called, parenchymatous neuritis—naked-eye changes rarely occur. Under the microscope many of the nerve fibres are found to have disappeared: there is increase in the connective tissue and hyperæmia. The vessel walls are usually thickened.

The mast cells of Ehrlich are seen, indicating excessive connective-tissue activity.

Diagnosis.—Multiple neuritis must be diagnosticated from diffuse or transverse myelitis, anterior poliomyelitis, locomotor ataxia, spinal meningitis, and hæmorrhage and Landry's paralysis. Practically, diffuse myelitis is the disorder from which it has oftenest to be distinguished. From this it is recognized first by

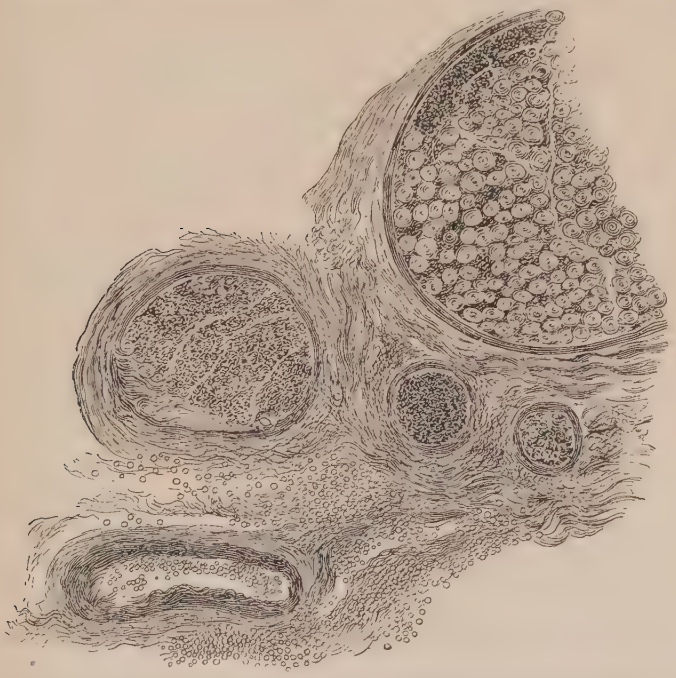


FIG. 45.—DIPHTHERITIC NEURITIS, CHIEFLY INTERSTITIAL (SIEMERLING); with secondary degeneration.

investigating the cause and onset. Neuritis begins more slowly and with sensory prodromata; it affects the legs and feet, especially the extensors, and if it ascends it skips the hips and trunks and attacks the forearms. There is more muscular atrophy than in myelitis; the knee jerks are absent. It progresses more slowly, and after four or eight weeks gradually regresses. Electrical degeneration reactions are more varied and decided. There are tenderness over the muscles and nerves and peculiar burning, darting pains. The cutaneous anæsthesia, if present, is not so extensive and complete, as a rule, while muscular anæsthesia is more decidedly marked. There is very

rarely involvement of the sphincters, or bed-sores. There may be belt-like constrictions felt round the extremities, but not around the waist. The gradual improvement of the paralysis and atrophy and eventual recovery confirm the diagnosis of neuritis. The presence of neuritis of the cranial nerves would strengthen the theory of a neuritis.

From poliomyelitis it is distinguished by the presence of pain and other sensory symptoms, the early fall in galvanic irritability, the age of the patient, and the etiology.

From locomotor ataxia, neuritis is distinguished by its more rapid onset, the presence of paralysis and atrophy of muscles, paresis, with degeneration reactions, and the absence of involvement of the organic centres.

Spinal hemorrhage usually leads soon to a secondary diffuse myelitis easily distinguishable from neuritis by the characters above given. Here there is also usually pain in the back. Spinal meningitis is associated with characteristic pain, tenderness, and stiffness along the back. Acute ascending paralysis in its typical form shows but very slight sensory disorders, and no wasting or change in electrical irritability.

The complication of multiple neuritis and myelitis or posterior sclerosis is possible, but is very rare. In the former case the ordinary symptoms of myelitis are added to those of neuritis. In locomotor ataxia there is often some nerve degeneration and occasionally neuritis. The nerve degeneration probably only causes slow atrophic changes and paresis; the neuritis causes pains, anæsthesia, skin eruptions, and local trophic disorders.

Prognosis.—Alcoholic multiple neuritis is a serious disease, because of its associated conditions. Nearly one-half of my patients have died, mainly because they continued the use of alcohol after paralysis appeared. They do not die of neuritis, but of alcoholism or of phthisis. Other forms of neuritis rarely cause death. The great majority recover almost completely. It may be from six months to two years before all symptoms disappear. The average time is about a year.

Treatment.—The patient needs, first of all, rest in bed. The limbs are often extremely tender and the patient's pains excruciating. To relieve these the legs may be painted with menthol and enveloped in cotton batting. In other cases flannels wrung out in hot water and renewed every two hours give relief. Internally phenacetin, antipyrine, salicylate of soda, may be given. In the early stages, salicylate of soda in doses of gr. xx. every two or three hours should be administered. If there is a great deal of depression from alcoholic poisoning, strychnia gr. $\frac{1}{60}$ q. 3 h. and aromatic spirits of ammonia, ʒ ss. q. 3 h., should be used. Nitrate of silver is also useful in the early stage.

After the acute stage is passed the labile galvanic current occasionally interrupted may be applied, 2 to 6 ma. for ten minutes three times weekly. Later, by the sixth week, the faradic current, massage, and careful exercise should be given. At this time or earlier (third week), strychnia, iodide of potassium, arsenic, and tonics may be given. In old cases in which a great deal of paralysis and contracture have occurred, forcible extension of the limbs, the use of splints and rubber muscles, are needed. With patience and perseverance the worst cases can eventually be brought to a complete recovery.

Complicating Forms of Neuritis and Neuritic Degeneration.—Neuritis and neuritic degeneration complicate many diseases, but they especially mark and modify subacute and chronic rheumatism, locomotor ataxia, diabetes, paralysis agitans, wasting diseases, and old age.

A *neuritic* degeneration almost always affects the nerves in the neighborhood of an old rheumatic joint. The chief result of this is to produce wasting and some paresis of the muscles moving the joint (Pitres and Vailtard). The process is a reflex atrophy (see Arthritic Muscular Atrophy).

In locomotor ataxia, parenchymatous degeneration is very often present. It does not produce the cardinal symptoms of this disease. It does, however, cause some of the anæsthesia, paræsthesia, muscular atrophy, skin dystrophies, and visceral crises.

In diabetes, the neuritis takes the form of the sensory type of multiple neuritis, and causes symptoms like those of locomotor ataxia. The patient has sciatic pains, burning or numb feet, loss of tendon reflex, ataxia. The upper extremities are rarely affected.

In Wasting Diseases and Old Age.—In various wasting diseases, such as phthisis, cancerous cachexia, long-continued fevers, marasmus, and in senility, a simple parenchymatous degeneration of nerves, with atrophy, occurs (Arthaud, Köster, Jappa). The symptoms caused by these changes are very slight. They contribute to the weakness and wasting. In old age, the atrophy of the nerves is one cause of the lessened sensibility and activity of the skin and its underlying muscles.

Morvan's Disease, Analgesic Paralysis with Whitlow.—(Neuritic type of syringo-myelia.)

This is a very rare disease, characterized by a slowly progressive paralysis and atrophy of the hands and forearms, with analgesia and painless whitlows.

It occurs usually in young or middle-aged males. Occupations, such as handling fish, which involve exposure and trauma, predispose to the disease.

The symptoms come on slowly, with, at first, severe pains in the arms and hands. The muscles of these parts gradually get

weak and atrophy. Anæsthesia and analgesia are present. Usually there is loss of pain and temperature, but not of tactile sense. Whitlows appear which are painless, and one or more of the terminal phalanges may necrose. There is usually a swelling and hard œdema of the parts. Both upper extremities may be affected, and sometimes the feet are also slightly involved. There is sometimes spinal curvature. Hysteria may complicate the affection. The course of the disease is very chronic, but there may be long periods in which the symptoms are quiescent.

Pathological examinations always show a neuritis of the parts involved in the disease. In some cases a syringo-myelia has been found.

There is, therefore, a Morvan's disease due to syringo-myelia and neuritis, and another type due to a neuritis alone.

The prognosis, so far as cure is concerned, is bad, but the disease may remain stationary a long time or improve.

The diagnosis is based on the combination of paralysis, atrophy, loss of pain and temperature but not of tactile sense, and whitlows.

Strychnine seems to have stopped the progress of the malady in one case. The treatment in general is only symptomatic.

FUNCTIONAL DISORDERS OF THE PERIPHERAL NERVES.

These are:

1. Motor forms: tremor, spasm, paralysis.
2. Sensory forms: neuralgia, paræsthesia.
3. Mixed neuroses.

1. TREMOR AND SPASM sometimes occur from peripheral nerve irritation. The cause is over-use of the limbs, and the symptoms are associated with others belonging to the occupation neuroses (see p. 468).

WAKING NUMBNESS (NIGHT PALSY, RECUMBENT PALSY).

This is a disorder characterized by a temporary paralysis of an extremity, with numbness, noticed on first waking, or after lying down for a time.

It is a rare disease and little is known of its cause. It occurs in adults and usually in the neuropathic. Sometimes evidence of weak heart or poor innervation of the vessels is present.

The *symptoms* are much like those caused by temporary compression of a nerve when the leg or arm go to sleep. The paralysis is temporary and there is no anæsthesia. It is often very obstinate.

The *treatment* consists in giving strychnine, mineral acids, and electricity, with occasional courses of bromides; iron and antirheumatics may be needed in some cases.

2. Sensory Neuroses. Neuralgia.

Definition.—Neuralgia is a functional disease characterized by pain in the course of a nerve or of nerves.

Forms.—Neuralgias may be idiopathic, *i.e.*, developed spontaneously, or symptomatic, due to some known toxic influence, reflex irritant, or organic disease acting on the nerve. Thus a central disease of the nervous system or a tumor pressing on a nerve may cause a symptomatic neuralgia. When, however, there is organic disease of the nerve itself, such as neuritis, the disease cannot be called neuralgia. It is often impossible to draw the lines absolutely.

Neuralgias are divided, in accordance with their cause, into the epileptiform, hysterical, reflex, traumatic, gouty, etc.

Neuralgias are also divided, in accordance with their anatomical location, into trigeminal, cervico-occipital, brachial, intercostal, lumbar, crural, sciatic, and visceral.

Migraine and headache are not classed among neuralgias.

Frequency.—Neuralgias of all kinds make up about ten per cent of the nervous disorders for which the neurologist is consulted. The most frequent form is the trigeminal; next in order come the sciatic, intercostal, cervico-occipital, lumbo-abdominal, brachial, and visceral neuralgias.

Etiology: Predisposing Causes.—Neuralgia never affects young children, and, leaving out migraine and headache, it is rarely met with before the fifteenth year. From that time until twenty-five the frequency very rapidly increases. About one-fourth occur between the ages of fifteen and twenty-five; the relative number then slowly decreases to the forty-fifth year, when there is a rapid fall. Neuralgia is very rare in old age.

Women are more affected than men in the proportion of five to three (analysis of 887 cases). In New York most cases occur in winter, next in the autumn. More cases occur in temperate climates and in wet and cold regions.

Hereditary influence, neurotic constitution, anæmia and debility, gouty and rheumatic diatheses, all predispose somewhat to the disease. The exciting causes can be included under the head of toxic agents, infections, exposure, over-exertion, emotional shock, and injuries.

Local irritation of nerves may excite neuralgia, direct or reflex in kind. Some help in recognizing the reflex origin of neuralgias and other pains may be gained from the accompanying diagrams.

Symptoms.—These will be described in detail in the chapters on the special forms.

The dominant symptom is, of course, pain. This pain is sharp, darting, boring, stabbing, or burning in character. It comes on in paroxysms of great intensity. In the intervals there may be no pain or it may be simply a dull ache. The pain runs along the course of certain nerves, it is not confined necessarily to them, but may be somewhat diffuse. Pain is increased or brought on by cold or heat, or pressure on the affected part.

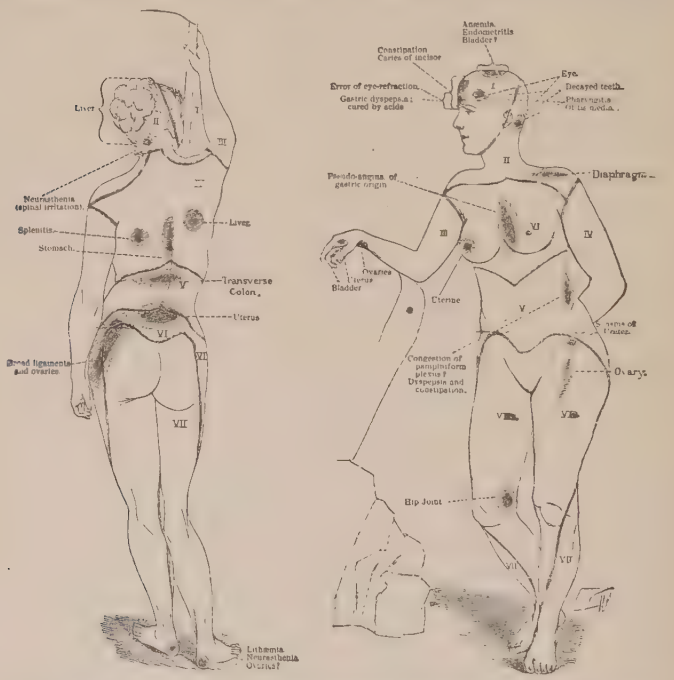


FIG. 46.—DIAGRAMS SHOWING THE DISTRIBUTION OF THE CEREBRO-SPINAL STRANDS OF NERVES AND THE LOCATION OF TRANSFERRED PAINS AND NEURALGIA.

	Strands of Cerebro-Spinal Nerves.	Distribution.
Area I.	Trigeminus, facial, etc.	Face and its orifices, anterior scalp.
Area II.	Upper four cervical.	Occipital region, neck.
Area III.	Lower four cervical and first dorsal.	Upper extremities.
Area IV.	Upper six dorsal.	Thoracic wall.
Area V.	Lower six dorsal except last.	Abdominal wall, upper lumbar, upper lateral thigh surface.
Area VI.	Twelfth dorsal, four lumbar.	Lumbar region, upper gluteal, anterior and inner thigh and knee.
Area VII.	Fifth lumbar and five sacral.	Lower gluteal, posterior thigh, leg.

The skin and nerves are sometimes tender or even exquisitely sensitive. Firm pressure, however, is usually not painful.

In about half the cases of long standing, *tender points* may be found which correspond to the exit of nerves from a bony canal or the substance of a muscle or fascia. In rare cases there is tenderness over the spine corresponding to the point where the affected nerves arise.

Besides feelings of pain, there is often a sense of numbness, coldness, tingling, or heaviness of the limb. Vasomotor, secretory, and trophic disturbances may occur; but when these are pronounced, one must suspect neuritis or an organic central disease. Muscular spasm is sometimes present; more rarely there is some muscular weakness.

The paroxysms of pain may intermit regularly; sometimes they come on every day, at the same hour. They are apt to be worse at night.

The attacks of the disease often run a course of several weeks or months, and in some forms they last for years.

Pathology.—Many cases that used to be called neuralgia are now known to be forms of neuritis, *e.g.*, sciatica and brachial neuralgia. Other forms are sometimes due to impaired nutrition of the nerves from an obliterating arteritis (tic douloureux); still others are due to the irritation of nerves from the diathetic poison of gout, rheumatism, and diabetes, or to extrinsic poisons, such as alcohol, lead, and arsenic. In these cases the sensory nerves of the nerve sheaths (nervi nervorum) are affected. There remain many cases in which the trouble shifts from one locality to another, or in which no special local or general irritation can be discovered. In these cases we assume that the pathogenic focus is the spinal ganglia or the sensory cells of the posterior horns of the spinal cord. Lesions and irritations of the sensory path above the sensory cells referred to rarely cause pain.

There are some forms of neuralgia which may be called "reminiscent" or "hallucinatory." The patient, who is an impressionable and sensitive person, has had a genuine cause for neuralgic pains; but this, after lasting some time, has ceased, while the painful impressions continue to remain in the cerebral cortex. The neuralgia is a morbid habit of feeling pain. Such neuralgias are promoted often by the use of morphine.

The Diagnosis.—This depends on the fact that neuralgic pains are shifting, paroxysmal, follow the course of nerves, are accompanied often with tender points and not accompanied with signs of organic nerve disease, such as paralysis and anæsthesia and tenderness over the nerve trunks. Thermic sensations of burning or coldness are rarely neuralgic, but are due to neuritis.

The prognosis is good, except for the reminiscent neuralgias,

the neuralgias of hysterical and neurasthenic persons, who are thoroughly anæmic, debilitated, and broken down morally and physically. The neuralgias of the degenerative period of life are also very obstinate.

The treatment will be discussed under special heads.

The Paræsthesic Neurosis (Paræsthesia, Numb Hands).

This is a not uncommon disorder, characterized by very persistent symptoms of numbness, formication, or burning, with little or no pain, and with or without motor weakness. It is a trouble allied to the *waking numbness* just described.

Etiology.—The disease occurs in adult life, and oftenest at or after middle life. Women are oftenest affected, and especially those of nervous temperament, with dyspeptic and rheumatic tendencies. The symptoms are not seen in children or in the aged. They are more marked in the warmer season, and women who wash a good deal are somewhat more subject to them. The disorder is more frequent in cities.

Symptoms.—The patients have feelings of numbness, prickling, heaviness, usually in both hands, extending a little way up the forearm. The sensations are, as a rule, diffuse, but may follow the distribution of a nerve, especally the ulnar. In other cases the numbness is sharply limited to the finger-tips or the second and third phalanges. There is no actual pain or tenderness, nor is there any anæsthesia, cutaneous or muscular. Instead of or with the numbness there may be burning sensations, especially felt in the palms or soles. There is often a slight degree of paralysis, but rarely any decided vasomotor or trophic changes. The symptoms exacerbate, being worse at certain periods of the day, usually in the morning. They may increase at night and prevent sleep.

The arms are oftener affected than the lower extremities; and the disease never takes a hemiplegic form, though one arm may be alone involved. The scalp and ears may be involved, though always in a minor degree, the patient complaining of sensations of heat, prickling, flushing, and vertex pressure. Sometimes the knee jerk disappears. The symptoms cause a condition of mental unrest and nervousness which add to the sufferings materially. Evidences of overwork, of dyspepsia and constipation, of anæmia, or of muscular rheumatism will generally be found. The urine is usually rather light in color and specific gravity (1010 to 1018), slightly cloudy, with excess of phosphates and occasional excessive discharges of urates.

Pathology.—The pathology is based upon the similarity of the symptoms to those in mild or incipient cases of multiple neuritis

and upon the effects of drugs in relieving symptoms. It is believed that the peripheral nerves are being irritated by some poison circulating in the blood. Co-operating with this are nerves naturally over-irritable, or made so by anæmia, hyperæmia, and exposure to cold.

Diagnosis.—This disease is to be distinguished from hysteria, neurasthenia, and central organic diseases of cord or brain. In hysteria the symptoms are less diffuse, regular and bilateral, while some anæsthesia is rarely absent. Paræsthesiæ are very common in neurasthenia, and in some cases here they are due, no doubt, to peripheral irritation. In neurasthenia, the paræsthesiæ are, however, generally in one extremity, less diffuse and as a rule more temporary. There is no motor weakness, and the head and spine are usually complained of more than the arms or legs. Waking numbness and night palsy are intermittent disorders, disappearing within a short time after waking or rising from a recumbent posture. The numbness that is felt with digiti mortui and Raynaud's disease is accompanied with spasm of the blood-vessels and whitening of the fingers.

Prognosis and Course.—The disease, if not treated, runs a course of several months, with some intermissions and relapses. It may disappear, to return the next year. It never progresses to any serious condition, and is in almost all cases eventually cured.

Treatment.—The patient should receive alkalies, with bromides, pepsin, iron, and strychnia. The faradic current, massage, and stimulating liniments are useful. Rest and removal from exciting causes, such as exposure, are also indicated.

3. Neuroses of Mixed Origin Affecting the Extremities (Acro-Neuroses).

There are certain nervous disorders which affect the extremities. They involve the motor, sensory, vasomotor, secretory, and trophic nerves in various degrees. They are sometimes of central and sometimes of peripheral origin; but as the pathology is little known, they are best given a clinical classification and placed under the head of peripheral diseases.

The acro-neuroses here considered are: Raynaud's disease and digiti mortui. Podalgia, luxation neuralgia. Erythromelalgia.

Symmetrical Angio-Neurotic Gangrene, or Raynaud's Disease. (Abortive Form Known as Digiti Mortui.)

Symmetrical gangrene or Raynaud's disease is a rare affection characterized by spasm of the vessels of the extremities, coldness, pallor, waxiness of fingers or toes, or by blueness, mottling, swell-

ing, pain, followed often by a dry gangrene of some of the fingers or toes.

Etiology.—The disease occurs usually in children and young adults. Women are affected oftener than men. Anæmia, and chlorosis, and neurasthenic states predispose to it. Malarial infection, acute infectious fevers, menstrual disorders, fright, occupations that lead to exposure, such as washing, are causative factors. Diabetes and syphilis are also put down as causes.

The disease comes on rather suddenly and affects oftenest two or three fingers of both hands. In its early and mild degree there is simply a coldness, numbness, and waxy pallor of the fingers. The skin looks shrunken. There is slight anæsthesia. They feel

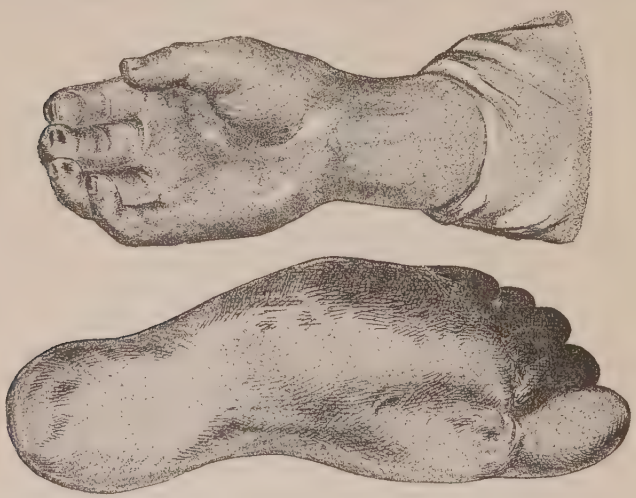


FIG. 47.—HAND AND FOOT IN THE GANGRENOUS STAGE OF RAYNAUD'S DISEASE.

as if dead. After a few hours this passes away, but returns again and may finally become an almost constant condition. Beginning in one or two fingers, it may finally involve all. The toes, tip of the nose, and ears may be similarly affected, though this is rare in the milder form. Exposure to cold, even slight, is the common excitant of this form of the trouble, which is commonly known as "digiti mortui," "dead fingers," or "local syncope."

In severer grades the fingers become blue, swollen, and there are burning sensations and much pain, but no anæsthesia. This condition is known as that of "local asphyxia," and it is usually followed by gangrene.

In the gangrenous stage small blisters appear on the distal phalanges, which fill with bloody serum, then dry up, and beneath

the scab ulceration begins, which is shallow and soon heals, leaving a scar. The process then stops. In very rare cases the whole tip of the finger or toe, including the bone, becomes involved. The process as stated may attack the ears, lips, tongue, and even parts of the trunk. Along with this gangrenous process there is often a hæmaturia.

The dead-finger trouble may last but a few days or weeks, or it may continue for months. The gangrenous stage lasts about three weeks. It lasts longer if it comes on in one finger after the other. The disease is one of months, and it is liable to recur.

The *diagnosis* must be made from senile gangrene, frost-bite, ergot-poisoning, alcoholic neuritis, endarteritis, and obstruction of nutrient vessels.

Pathology.—A neuritis has been found in some cases of so-called Raynaud's disease, but this is secondary. In a few other cases there has been found an obliterating endarteritis (Jacoby).

The trouble is in some cases apparently functional and due to the combination of an over-sensitive nervous system and some irritant, such as impoverished blood, malaria, or other toxic agent, which causes spasm of the peripheral vessels.

Prognosis.—The cases usually get well. In only the rarest instances has death occurred, and then from some complication.

Treatment.—Galvanism to the spine and limbs, warm applications, anodynes, tonics, are indicated. Nitroglycerine, the iodides, chloral may be tried. No specific is known.

Pains in the Feet of Various Origin (Podalgia).

1. Pains of diathetic, toxic, or neuralgic origin.
2. Pains of reflex origin.
3. Pains of mechanical origin.

1. Pedal pains occur in gout, rheumatism, lithæmic states, syphilis, tabes, chronic alcoholism, and diabetes. The pains of latent gout, according to Anstie, are generally located in the dorsum of the foot. Those of chronic alcoholism have been observed to occur in the wrists and ankles. Those of lithæmia are usually in the heels. In syphilis a node can generally be discovered. A differential diagnosis is therefore possible.

Erythromelalgia (*red neuralgia of the feet, congestive neuralgia*) is a disease affecting the feet chiefly, and characterized by burning pains and congestion of the parts.

The disease occurs usually in men in middle life, after a low fever or severe physical exertion afoot.

The disease begins in the ball of the foot or the heel with a pain, which is worse at night. It increases until nearly the whole sole in the distribution of the plantar nerve is involved, and the

pain, though worse at night, is almost continuous. It is much increased by exertion, the feet become very tender, and standing or walking is most painful. Meanwhile there has developed with the pain a flushing of the part upon exertion. In bad cases the parts most affected are continuously marked by a dull, dusky, mottled redness.

The hands may be slightly affected. Slight injuries may cause blisters and even ulcerations. The congestion usually disappears in the horizontal position, and this also relieves the pain. The symptoms are worse in warm weather. The disease is very chronic and, though not dangerous to life, makes life very miserable.

Pathology.—In the cases of erythromelalgia as described by Mitchell and others, there is: (1) A vasomotor neurosis; (2) a plantar neuritis; and (3), in rare instances, spinal cord disease.

The *diagnosis* must be made from alcoholic and gouty paræsthesia, podalgia, local disease of bone and ligaments and from reflex pains.

Treatment.—Elevation of the feet and applications of cold give temporary relief. Faradization has sometimes given help; oftener it has not. There is nothing known which gives permanent relief. The physician must rely upon rest, bandaging, cold, anodyne applications, hydrotherapy and tonics. The salicylates, turpentine, and mineral acids with strychnia may be given.

2. Reflex or referred pains in the feet are very common. The cause is usually some form of irritation of the genito-urinary tract, such as gonorrhœa, stricture of the urethra, vesical calculus, cystitis, or renal calculus. It may be a misplaced or pregnant uterus.

3. Pains of mechanical origin:

(*a*) Morton's neuralgia.

(*b*) Tarsalgia, or policeman's disease.

(*a*) MORTON'S NEURALGIA, so called, is a neuralgia affecting the metatarso-phalangeal joint of the third and fourth toes, and is due, it is thought, to a slight luxation, with consequent pressure on a digital branch of the external plantar nerve. It sometimes affects other toes, however. It is not always due to a luxation. Incipient flat-foot may cause it, and I have seen a typical case in a pregnant woman, disappearing after confinement. The trouble occurs generally in women, and if there is a luxation the cause is external injury or shoe-pressure. Treatment is not very satisfactory. It should be directed to giving rest to the foot, and the avoidance of lateral pressure on the joints by wearing a broad-soled shoe with support to the arch of the foot. Support may also be given by a broad flannel bandage. Amputation of the toe has cured one case.

(*b*) TARSALGIA (*policeman's disease*) is a neuralgic affection,

due probably in most cases to an incipient flattening of the foot and stretching of the plantar ligaments. Some have ascribed it to a deep-seated contusion of the adipose cushion covering the os calcis. Probably the condition varies somewhat in different cases. It is observed in persons who have been in the habit of going barefoot, and have then gone into the army or taken civil positions obliging them to stand or walk a great deal.

It occurs in the policemen of Paris, and but few cases have been seen in this country. This is due to the difference in the shoes and in the gait of American and Parisian guardians of the peace. The name tarsalgia was given by Duchenne.

Treatment, medical or surgical, seems to do little for the disorder, which is very chronic. Patients are better in cold weather, and when resting the feet. Leeches, the cautery, the iodides, and broad shoes with rubber heels are serviceable.

Tumors of Nerves.

These consist of:

1. Nerve hyperplasia.
2. True neuromata. } Single. { Benign. Malignant.
3. False neuromata. } Multiple. { Benign.

1. *Hyperplasia* or hypertrophy of nerve trunks is very rare. Generally the increase in size is, in fact, due to increase of the interstitial connective tissue. Sometimes there is an increase in the number of fibres and thickening of the myelin sheath.

2. *True neuromata* are also very rare, and occur almost exclusively on spinal nerves. In some there is an increase in medullary fibres; in others only an increase of non-medullated fibres, *i.e.*, only the axis cylinders and neurilemma increase. They occur either singly or multiply. *Multiple neuromata* are generally of a neuro-fibromatous character.

Neuro-fibromata when multiple may affect the subcutaneous nerves and form growths known as *fibroma molluscum*.

Nerve fibres united in a mass by hyperplastic connective tissue form what are called *plexiform neuro-fibromata*. Multiple neuromata may be true neuromatous growths.

True neuromata are usually small, ranging from 1 cm. (two-fifths of an inch) to 6 cm. in diameter. They may be much smaller or larger.

Neuromata are usually few in number, or at least there is only a local multiplicity of tumors. Multiple (true) neuromata may, however, be very numerous. Gowers estimates in one case that as many as one thousand were present. Even larger numbers have been observed.

3. *False Neuromata.*—This term is applied to the various nerve tumors in which a fibroma, myxoma, glioma, sarcoma, carcinoma, or syphiloma grows upon or in the nerve. Fibro-neuroma is the most common form; glio-neuroma has been observed on the auditory nerve. Syphiloma occurs only on the intra-cranial or intra-spinal nerves. Carcinoma of nerves may be primary, but is generally secondary, and is of scirrhous or medullary type, rarely the colloid. Leprous neuritis sometimes forms neuro-fibromatous swellings.

Traumatic fibromata are simply small false neuromata situated subcutaneously on the ends of the sensory nerves. They vary much in histological structure.

Malignant Neuromata.—A few cases, about thirty in all, have been observed of multiple malignant neuromata. Trauma and hereditary influence are the etiological factors. The great nerve trunks are oftenest affected, the median, sciatic, and crural ranking first. The tumors start from the perineurium: they are at first spindle-shaped, and may grow very large. Sarcomatous cells are oftenest found in them: but they may be myxomatous, fibromatous, or mixed.

Etiology.—Three general causes exist for the production of neuromata, viz. 1. A hereditary or a neuropathic predisposition: this tends to cause the true, the multiple, and the plexiform neuromata. 2. Injuries, surgical operations: these cause especially the fibro-neuromata of which the *amputation neuroma* is an example. 3. Diathetic, *e.g.*, tubercular, influences and whatever produces the various tumor formations, sarcoma, carcinoma, form the third etiological factor. Neuromata of the plexiform type are often congenital. Multiple neuromata may develop early in life. Men are far more subject to multiple neuromata than women.

Symptoms.—Neuromata often cause no symptoms. Perhaps the most frequent evidence of their presence, however, is pain and some tenderness. The pain is exacerbating, and may be stopped sometimes by pressure on the nerve above the tumor. Paræsthesia, anæsthesia, paralysis, and reflex spasm may be present. Some forms of intractable headache are possibly due to multiple neuromata. Multiple and plexiform neuromata cause symptoms less often than a single larger neuroma. A neuroma on the pneumogastric or other splanchnic nerve may cause severe symptoms. Multiple (true) neuromata may last for years and cause no serious inconvenience. Malignant neuromata cause always such symptoms as would naturally follow irritation and compression of a nerve.

The *diagnosis* of neuroma can only be certainly made when the tumor can be felt. In other cases, by exclusion a fairly certain conclusion may be reached.

True neuromata are often multiple; the false rarely. Idiocy, heredity, neuropathic constitution, scrofula, would all favor the view of the disease being a true neuroma.

The *treatment* is essentially surgical. Internal medication and external applications are of little value. Strong galvanic currents, mercury, and the iodides in large doses may be tried in true neuromata and fibro-neuromata.

Both true and false neuromata may return after extirpation.

CHAPTER VII.

DISEASES OF THE SPECIAL NERVES.

THE peripheral nerves are the cranial, the spinal, and the offshoots of the cranial and spinal, known as the sympathetic. I shall take up first the cranial nerves.

THE CRANIAL NERVES.

These consist of twelve pair, viz.:

I. The olfactory. II. The optic. III. The motor oculi. IV. The patheticus or trochlear. V. The trigeminus. VI. he abducens. VII. The facial. VIII. The auditory. IX. The glossopharyngeal and nerve of Wrisberg. X. The vagus. XI. The accessory vagus or spinal accessory. XII. The hypoglossal.

The first two are not strictly nerves, but rather parts of the brain.

The remainder are analogous to spinal nerves, and arise from the mid and hind brain.

ANATOMY AND PHYSIOLOGY.—I. The *olfactory nerve* consists: 1. Of peripheral fibres which are distributed to the superior and middle turbinated bones and upper part of the septum. They are connected with olfactory cells lying in and beneath the mucous membrane. They are about twenty in number on each side. They are non-medullated. They pass up through the cribriform plate of the ethmoid bone and enter the olfactory bulb (Fig. 48).

2. The olfactory bulb is in man a rudimentary organ containing a few nerve cells, peculiar bodies, perhaps of neuroglia tissue, called glomeruli, connective tissue, and nerve fibres. It is analogous to a spinal ganglion or to a nucleus of origin of a nerve in the cord or medulla.

3. The olfactory tract, wrongly called a nerve, is composed of cortical nerve cells and of nerve fibres. It has three roots, the lateral, middle, and mesial. In man, only the lateral is of importance. This passes along the margin of the anterior perforated space to the uncinate gyrus, hippocampus, and cornu ammonis. Some fibres also pass inward into the anterior commissure, connecting the tract and cortex of the opposite side (Fig. 48). 4. The fourth

portion of the olfactory apparatus is the uncinate gyrus, hippocampus, and cornu ammonis, and this part is the cortical seat of smell. Further connections by the fimbria, fornix, corpora mammillaria, and optic thalami are thought to exist (Hill); also direct connections with the basal ganglia. The olfactory fibres do not decussate like the other nerves.

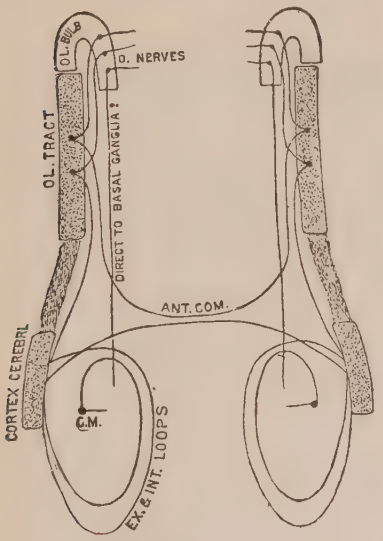


Fig. 48.

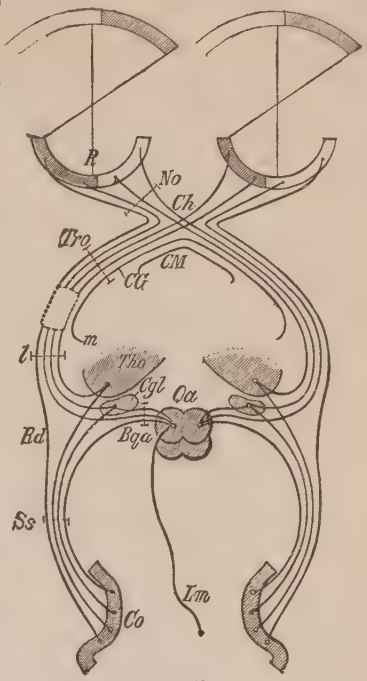


Fig. 49.

Fig. 48.—Diagram of Olfactory Nerves and Connections.

Fig. 49.—The Optic Nerve and its Central Connections (modified from Obersteiner). R, Retina, dark on the side connected with left hemisphere; *No*, optic nerve; *Ch*, chiasm; *Tro*, optic tract; CM, Meynert's commissure; CG, Gudden's commissure; *Tho*, thalmus; *Cgl*, corpus genic. ext.; *Qa*, corpor. quad.; *Ss*, optic radiations; *Co*, occipital cortex; *Lm*, mesial lemniscus. (The shading of the retina and visual field should just reach the vertical lines.)

The sense of smell is rudimentary in man, yet it is still the sense by which we can appreciate the most attenuated matter; for the trillionth of a grain of mercaptan, which awakens a sensation in the mind, has weight and dimensions so infinitely minute as to be quite beyond the power of the imagination to grasp. According to Valentin, we can perceive $\frac{1}{1200000}$ of a grain of oil of roses. According to Fischer and Penzoldt, one can perceive $\frac{1}{27000000000}$ of a grain of mercaptan. I have found that one can perceive the odor from 4 cm. of a solution of oil of cloves 1

to 100,000. Matter to be perceived as odor must be in a gaseous form. Odorous sensations co-operating with taste sensations form "flavor." Variety in odorous sensations depends probably upon the rapidity of molecular vibrations as in the case of light; and substances having similar relations in vibration have similarity in odor (Haycraft). Males have a more delicate sense of smell than females (Nicolls and Bailey). The keenness of the olfactory sense is lessened in the insane and criminal classes.

II. The *optic nerve*, so called, is really a central tract of the brain, like the olfactory nerve.

Traced from without inward, we find the following parts:

1. The rods and cones, with the filaments connecting them to the ganglionic cells of the retina, really form all that there is of a peripheral optic nerve. These rods and cones number about 3,360,000.

2. From the ganglionic cells of the retina, fibres collect and pass into the cranial cavity as the optic nerve. This nerve contains about 438,000 fibres. They are smaller than other nerve fibres, and are distinguished from them by having a neuroglia imbedding substance and by having no neurilemma.

3. The optic nerves pass to the optic chiasm, where about one-third of the fibres cross, in man. In lower animals the decussation is greater. After leaving the chiasm, the fibres form the *optic tract*. This tract curves up and back around the crus cerebri, and divides into a lateral and mesial root.

4. These roots connect with the external geniculate body, the anterior tubercles of the corpora quadrigemina, and the posterior ganglion of the thalamus or the pulvinar. These ganglia are called the primary optic centres. Through the anterior tubercles of the corpora quadrigemina, and by other means, the optic nerve is connected with the oculomotor nerve, and thus reflex movements of the pupils, lids, and eyeballs are brought about (Fig. 49).

5. From these primary optic centres, fibres enter the posterior part of the internal capsule, curve up and back toward the occipital lobes, forming the optic radiations of Gratiolet (*Ss*).

6. They are finally distributed to the cortex of the occipital lobe, and in man chiefly to the cuneus and the upper end of the first occipital gyrus.

It will be seen that each retina is connected with the occipital lobe of both hemispheres. It is further known that the outer or temporal half of each retina is connected with the occipital lobe of the opposite hemisphere, and the inner or nasal half of each retina with the occipital lobe of the same side. Further, the upper part of each retina is connected with the lower half of the cuneus, and *vice versa* (Fig. 49).

Other connections exist by which the optic centres on the two

sides are connected with each other and with other cranial nerves in the medulla.

The optic nerve is a nerve of special sense of vision and has no other function except that of an excito-reflex character.

III., IV., VI. The *motor nerves of the eye* are:

(*a*) The third or oculo-motorius, supplying the internal superior and inferior recti, inferior obliquus, the levator palpebræ, the ciliary muscle, and constrictor of the iris.

(*b*) The fourth or trochlearis, supplying the superior oblique.

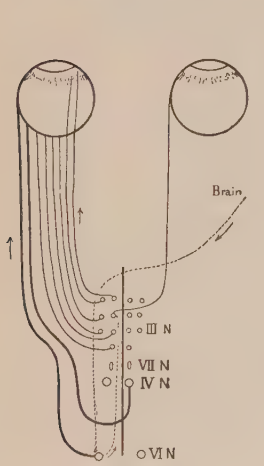


Fig. 50.

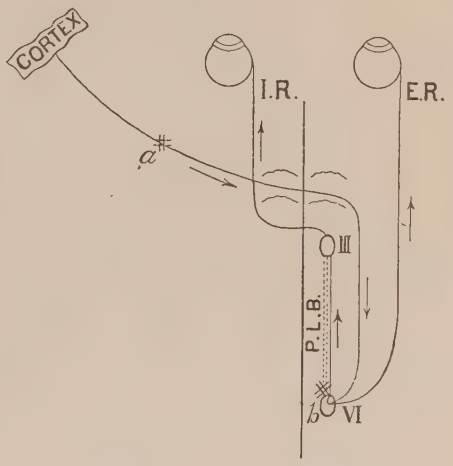


Fig. 51.

Fig. 50.—Diagram showing the Arrangement of the Nuclei of the Motor Nerves of the Eye, and the Decussations of the Fourth and Internal Rectus Branch of the Third Nerve.

Fig. 51.—Diagram showing the Probable Relations of the Nuclei of the Sixth and of the Internal Rectus Branch of the Third to the Brain.

(*c*) The sixth or abducens, supplying the external rectus.

(*d*) The sympathetic, consisting of fibres from the upper cervical nerves to the dilators of the iris, to its blood-vessels, and to Müller's muscle.

2. Motor fibres from the nucleus belonging to the third nerve and running out with the fibres of the seventh (Mendel), supplying the orbicularis palpebrarum.

The third and fourth nerves arise from a series of nuclei in the floor of the aqueduct of Sylvius. They leave the brain at the anterior edge of the pons. They run in the cavernous sinus and enter the orbit through the sphenoidal fissure.

The sixth nerve arises from a nucleus in the floor of the fourth ventricle. It emerges at the posterior edge of the pons, runs in

the cavernous sinus, and enters the orbit through the anterior lacerated foramen.

The nuclear gray matter from which these nerves arise is made up of a series of nests of cells, and each pair supplies a different set of muscles of the eye, as shown in the diagram.

The nucleus of the sixth lies farther back in the floor of the medulla, but it belongs to the same serial deposit of gray matter, and represents the continuation of the anterior horn of the spinal cord (Fig. 51).

The motor nerves of the eye, third, fourth, and sixth, are closely connected with each other and other nerves by a long commissure, the *posterior longitudinal bundle.*

The fibres of the third and fourth nerves pass to their nuclei on the same side, then decussate and pass up in the inner part of the crusta to the lower part of the central convolutions of the cortex. A few fibres decussate and enter the nuclei of the opposite side. They are connected with the internal rectus nucleus.

The fibres of the fourth nerve almost entirely decussate, running forward a long distance before they finally reach their nuclei. Thus it appears that the abducens is the only cranial nerve except the optic which largely decussates before reaching its nucleus. However, those fibres of the third which supply the internal rectus also decussate, as already stated.

The arrangement of the nuclei is believed to be about as follows, the upper on the list being anterior:

Median line.

III. N.	Sphincter iridis, Ciliarius.
	Levator palp., Rect. int.
	Rectus superior, Rect. inf.
	Obliquus inf.
IV. N.	Obliquus superior.
VI. N.	External rectus.

In order to understand the peculiarities of eye palsies, to be described later, the relations of the sixth to that nucleus of the third nerve which innervates the internal rectus must be understood. In turning the eyes to one side, these two nuclei and their nerves act together, causing the external rectus of one eye and the internal rectus of the other to contract at the same time. The impulse from the brain which does this acts first upon the sixth, and through this upon the internal rectus nucleus of the same side. The fibres from this nucleus cross and emerge in the third nerve fibres of the opposite side to the sixth nerve nucleus. This can be better understood by the diagram, Fig. 51.

Thus lesions in the brain (at *a*) cause paralysis of the sixth nerve of the opposite side and internal rectus of the same side. The eyes turn toward the side of the lesion.

Lesions in the pons (at *b*) cause paralysis of the sixth on the same side and internal rectus nucleus of the opposite side. The eyes turn away from the side of the lesion.

The eye muscles move the eyeball in the following way:

The superior rectus elevates the eyeball.

The inferior oblique rotates out and up.

The inferior rectus depresses the eyeball.

The superior oblique rotates out and down.

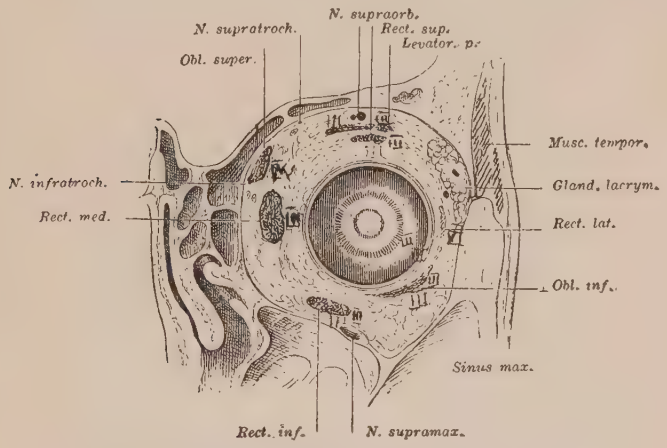


Fig. 52.—The Attachment of the Eye Muscles to the Globe (Merkel).

The superior and inferior oblique, acting together, rotate inward.

The external rectus rotates outward.

The internal rectus rotates inward.

The rectus internus, rectus superior, obliquus inferior, rotate upward and inward.

The rectus internus, rectus inferior, obliquus superior, rotate downward and inward.

The rectus externus, rectus superior, obliquus inferior, rotate out and up.

The rectus externus, rectus inferior, obliquus superior, out and down.

The movements of the eyeball are made by the simultaneous action of several muscles. Most of them act as their names indicate. But the oblique muscles help to depress and elevate, and

then help to rotate in or out according as the internal or external rectus is acting.

The cortical centres for the eye muscles are not positively known. Lesions in the inferior parietal lobule sometimes cause paralysis of the third nerve. Lesions of the posterior part of the prefrontal lobes also sometimes cause eye palsies, especially conjugate deviation.

As the optic nerve is the special sensory nerve of the eye, so the third, fourth, sixth, and part of the seventh nerves are the motor nerves.

By means of the optic nerve and its receptive and refractive apparatus, the form, color, movement, and, to some extent, relations and distance of objects are determined. The motor nerves

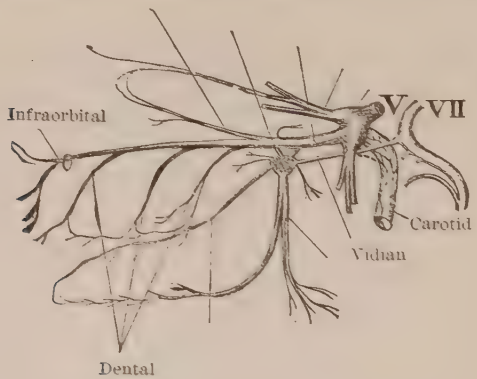


FIG. 53.—THE SUPERIOR MAXILLARY DIVISION OF THE TRIGEMINUS (YOUNG-BAKER).

adjust the eye to near and distant objects, inform us as to size and distance, and enable us to follow moving objects and to shift the gaze readily. They also assist in protecting the eye against injury.

V. *The trigeminus* or fifth nerve is one of the most extensively distributed and most delicately sensitive nerves of the body. Its sensory branches represent the atrophied and lost sensory roots of the third, fourth, sixth, seventh, and twelfth cranial nerves (Gaskill).

It has a most extensive primary origin by three principal roots: (1) The ascending, which begins in the upper part of the cervical cord at the level of the second cervical segment; (2) the descending, which rises in the gray matter about the aqueduct of Sylvius as high up as the anterior corpora quadrigemina; (3) the middle, which arises from a nucleus in the pons near the level of the

nerve's exit. The ascending root is mainly sensory; the descending, sensory and trophic; the middle, motor and sensory.

The gray matter from which the trigeminus arises reaches the whole length of the pons and medulla, and is coextensive with the origin of all the other cranial nerves (Fig. 57). Hence the frequency with which its disorders are complicated with those of these nerves. Its cortical origin is probably in the lower part of the pre- and post-central convolutions.

The trigeminus supplies sensation to the face, nose, the frontal and maxillary sinuses, the teeth, the palate, tongue, and part of the upper pharynx; also to the scalp as far back as the vertex

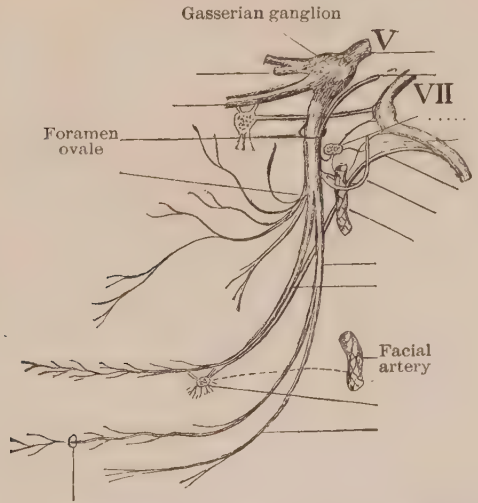


Fig. 54.—The Inferior Maxillary Division of same (Young).

and to the external auditory meatus. It gives sensation also to the anterior three-fourths of the dura mater, the falx, and probably the tentorium. The pia and arachnoid are not sensitive. The posterior fossa and the occipital part of the dura mater are supplied by the vagus. The trigeminus also supplies the above-named parts with trophic, vasomotor, and secretory fibres. The vasomotor fibres are brought to it from the medulla and cervical spinal cord via the sympathetic; the secretory fibres have the same origin. Both are reinforced by the four ganglia with which this nerve are in close connection. An exception is to be made of the lachrymal fibres which are brought by the motor nerves of the eyeball.

The trigeminus supplies motion to the muscles of mastication, viz., the two pterygoids, the temporal, masseter, mylo-hyoid, and anterior belly of the digastric.

VII. *The facial nerve* has its primary origin in a single nucleus deeply situated in the lower part of the pons. It belongs to the same series of nuclei as the motor nuclei of the vagus, glosso-pharyngeal, and of the spinal accessory; in other words, it is a prolongation of the lateral horn of the spinal cord. It has not a nucleus common to it with the sixth, as is usually stated. Those fibres of the nerve which go to the orbicularis, however, appear to come from a nucleus in the third nerve series and to reach the knee of the facial by the posterior longitudinal bundle (Mendel). The deep fibres of the facial take a tortuous course, passing inward, dorsally, then curving down and out around the nucleus of the sixth nerve.

The cortical origin of the seventh is in the lower part of the central convolutions, especially the precentral. The fibres pass down in the crusta at the inner side of the pyramidal or motor tract. They decussate and reach the nucleus. The nerve has its exit at the posterior edge of the pons, external to the sixth nerve. It then has to take a long course through a bony canal, during which it receives taste fibres from the second branch of the trigeminus (or the glosso-pharyngeal). These fibres leave the nerve as the chorda tympani, and join the lingual branch of the fifth nerve to supply taste to the anterior two-thirds of the tongue.

The facial nerve supplies motion to all the muscles of the face; to the stapedius, stylo-hyoid, buccinator, and platysma myoid. It also contains trophic and secretory fibres. It does not supply, however, the muscles of mastication.

The taste fibres of the facial nerve apparently come in many cases from the second branch of the fifth via Meckel's ganglion, the large superior petrosal nerve, and geniculate ganglion. In other cases they come from the glosso-pharyngeal nerve via the ganglion petrosum, Jacobson's nerve, tympanic plexus, and geniculate ganglion. Some think that the intermediary nerve of Wrisberg, which arises in the upper part of the glosso-pharyngeal nucleus and connects with the geniculate ganglion, carries taste fibres to the facial.

VIII. The *auditory* or eighth cranial nerve has two different parts. One portion passes to the cochlea and utricle and saccules; it has to do with the sense of hearing; the other goes to the semicircular canals, and has to do with that sense by which we appreciate position of our body and its relations to space. The eighth nerve is thus an auditory and a space-sense nerve.

The auditory fibres enter the medulla by two roots, a lateral or posterior and a median or anterior. The lateral root has mainly auditory fibres. The space-sense fibres enter chiefly by the median root. These roots are connected with three nuclei, viz.: (1) The chief nucleus (dorsal, central, inner nucleus); (2) the

large-celled nucleus (Deiter's); and (3) the accessory nucleus (ventral, anterior, lateral). The chief nucleus (1) is a large mass of gray matter composed of small nerve cells and lies superficially just beneath the floor of the fourth ventricle. The large-celled nucleus (2) lies to the outer side of and below it. The accessory nucleus (3) lies in the substance of the lateral root, and between it and the median root. The lateral root is the one coming chiefly from the cochlea, and is, as stated, mainly a nerve of hearing. It is connected most extensively with the accessory nucleus, but also with the other nuclei.

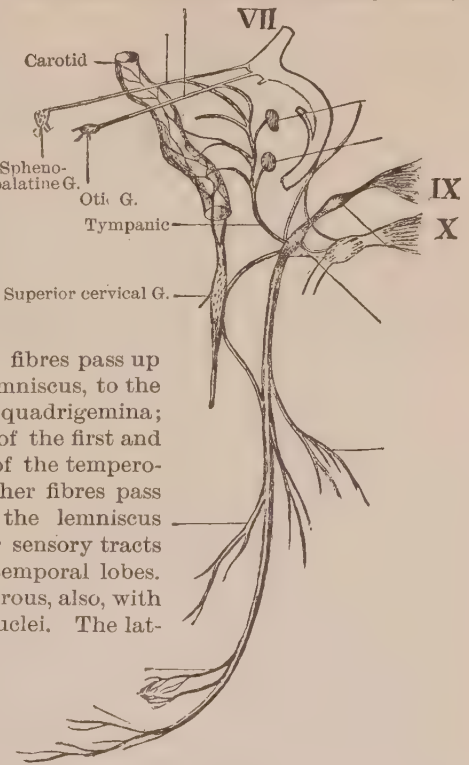


Fig. 55.—Glosso-Pharyngeal Nerve (Young).

Through the accessory nucleus, it connects by a few fibres with the superior olives mostly of the opposite side; thence fibres pass up through the lateral lemniscus, to the posterior corpora quadrigemina; thence to the cortex of the first and second convolutions of the tempero-sphenoidal lobes. Other fibres pass up directly through the lemniscus and the tegmental or sensory tracts to the cortex of the temporal lobes. Connections are numerous, also, with other cranial-nerve nuclei. The lateral root also sends fibres to the chief nucleus and (via the striæ acusticæ) into the raphe, and thence to the formatio reticularis and sensory tract. These fibres go also to the posterior tubercles of the corpora quadrigemina and thence to the cortex of the temporal lobe.

The median root is connected chiefly with the large-cell nucleus (2), but also with the chief nucleus (1). From these nuclei fibres pass up in the posterior cerebellar peduncle through the direct sensory tract of the cerebellum to the roof nucleus or the emboliform and globose nuclei.

IX. The *glosso-pharyngeal* nerve arises from three nuclei in

the medulla: (1) The sensory or perhaps visceral nerve cells analogous in character to those of the column of Clark in the spinal cord. (2) A true sensory nucleus consisting of a long, thin deposit of gray matter continued from the posterior horn of the spinal cord, and associated with a strand of nerve fibres called the ascending root of the vagus and pharyngeal nerves or respiratory bundle. (3) A motor nucleus which is called the nucleus ambiguus, and is a continuation of the lateral horn of the spinal cord (Hill, Gaskill).

The nerve, therefore, has motor, sensory, and probably visceral fibres. It supplies general sensation to the tympanum and pharynx (in connection with the vagus) and upper part of the larynx; special sensation of taste to the posterior third of the tongue, and motion to the pharyngeal muscles in connection with the vagus.

Its cortical representation so far as taste is concerned is in the hippocampal gyrus. The nerve gives very sensitive reflex fibres to the pharynx and is important in the reflex act of deglutition; it also carries sensations of nausea from pharyngeal irritation.

X. The *vagus nerve* arises from three nuclei in a way entirely similar to that of the glosso-pharyngeal.

1. The sensory and visceral nucleus.

2. The motor nucleus, called the nucleus ambiguus, which is a prolongation upward of the lateral horn, and is common to it and the glosso-pharyngeal.

3. The sensory nucleus, which gives off the fibres of the ascending root of the glosso-pharyngeal.

The nerve is closely connected with the glosso-pharyngeal above and with the accessory (eleventh) nerve below.

The accessory portion of the spinal accessory is practically a part of the vagus, and both in mode of origin and function resembles it closely. It arises from a nucleus which lies in the floor of the fourth ventricle, and is continuous brainward with that of the vagus. It consists of small cells giving origin to fibres that are small in size and have sensory, vasomotor, visceral motor, secretory, trophic, and excito-reflex functions.

The vagus and accessory part of the eleventh together have an extraordinarily wide distribution and diversity of function.

1. First they contain motor, inhibitory, and vasomotor fibres. These fibres go to the pharynx, larynx, trachea, and bronchi; to the œsophagus, stomach, small intestines, and spleen.

2. Sensory fibres, which go to the occipital and transverse sinuses and dura mater of the posterior fossa, to the external auditory meatus in part, to the pharynx, larynx, and trachea, and to the œsophagus.

3. Excito-reflex fibres, which go to the lungs and heart,

stomach, and to other organs mentioned as supplied by the vagus with sensation.

These reflex fibres stimulate or inhibit the vasomotor centre, the respiratory rhythm, and the cardiac rhythm. They also excite reflexly deglutition and respiratory movements.

The secretory fibres go to the respiratory tract, œsophagus, stomach, and pancreas and small intestines.

Cardio-inhibitory fibres go to the heart, while reflex accelerating fibres and inhibiting fibres go to the lungs. The accessory nucleus supplies the laryngeal adductors and the cardio-inhibitory fibres.

XI. The *spinal part of the spinal accessory* arises from the lateral horn and outer part of the anterior horn of the spinal cord. Its fibres of origin extend down as far as the fourth or fifth cervical roots. The fibres unite in the cranium and pass out through the posterior lacerated foramen in the same sheath as the vagus. After their exit from the skull, they divide into an internal and external part. It is the external branch which contains the fibres of spinal origin. The nerve receives some sensory fibres from the first and sometimes from the second cervical nerve. The terminal branches receive motor fibres from several cervical nerves. The spinal accessory contains large and small or visceral fibres. The spinal part contains only the large fibres.

Fig. 56.—The Vagus and Spinal Accessory Nerve (Young).

The spinal accessory supplies the sterno-cleido-mastoid almost exclusively, but only the upper part of the trapezius; the rest of this muscle is supplied by the cervical and dorsal nerves. The sterno-cleido-mastoid, when innervated, draws the chin up and over toward the opposite side.

The upper fibres of the trapezius draw the head back slightly and down toward the same side.

Physiologically the spinal part of the accessory nerve is one of the motor cervical nerves: the accessory or medullary portion is part of the vagus, and has visceral and sensory as well as motor functions.*

XII. The *hypoglossal nerve* arises from a long and large nucleus lying in the lower part of the floor of the medulla near the

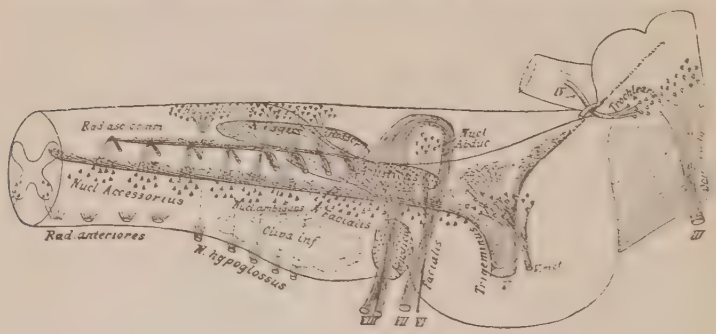


Fig. 57.—Longitudinal Section showing the Relative Position of the Cranial Nerve Nuclei (Edinger).

median line and to the outer and ventral side of the central canal. The nucleus is a continuation upward of the anterior horns of the spinal cord and is homologous with the sixth, fourth, and third nerve nuclei higher up (see Fig. 57). It reaches below as far as the decussation of the pyramids and above as far as the glosso-pharyngeal nucleus. A second small-celled nucleus lies just beneath the nucleus proper. Its cortical representation is in the lower end of the central convolutions, to which it is connected by fibres that pass into the raphe and thence to the anterior pyramids. Its fibres pass out between the olivary body and the anterior pyramid. At its origin it is a purely motor nerve; it receives a few sensory fibres from the cervical nerves and the vagus. It supplies the following muscles:

* Dees thinks that the spinal origin of the eleventh is continuous above with the twelfth, not with the anterior nucleus of the tenth. He denies that the medullary nucleus sends fibres to the larynx.

The internal branch of the eleventh sends motor fibres to the rectus posticus (E. Remak).

Intrinsic muscles of the tongue: superior and inferior longitudinal and transverse. The extrinsic muscles of the tongue: hyoglossus, genio-hyoglossus, and styloglossus. (The palato-

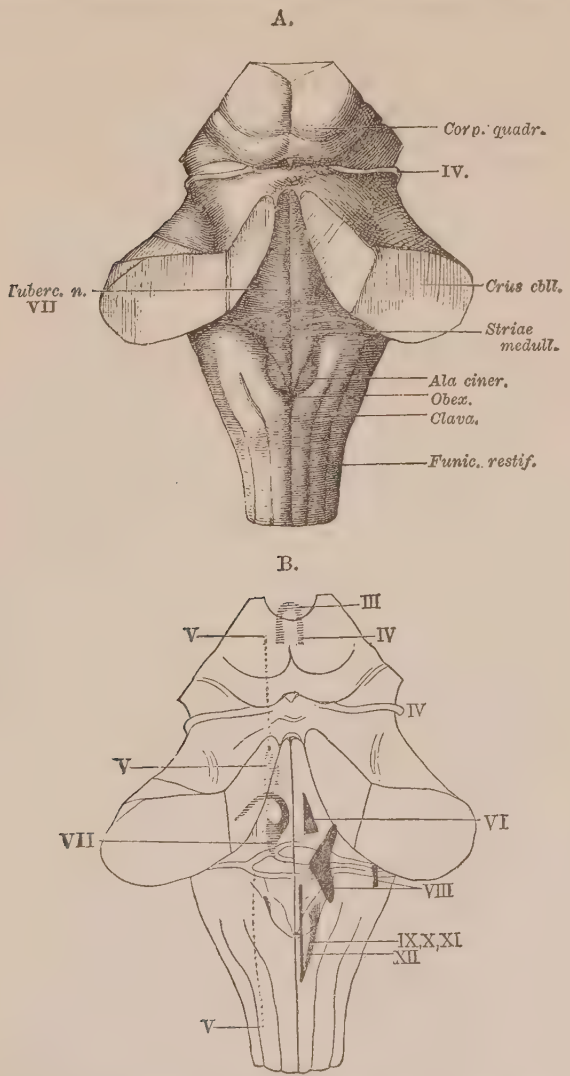


FIG. 58.—DIAGRAMS SHOWING THE POSITION OF THE CRANIAL NERVE NUCLEI AND THE FLOOR OF THE FOURTH VENTRICLE (MERKLE).

glossus and linguals are supplied by the fifth and seventh cranial nerves respectively.) The depressors of the hyoid: the thyro-hyoid and, with the cervical nerves, the sterno-hyoid and sterno-thyroid. The elevator of the hyoid: genio-hyoid. It is also thought to send fibres to the oral muscles (Tooth).

The hypoglossal nerve is concerned in the movements of the tongue and in fixing or depressing the hyoid in mastication and deglutition. When diseased, therefore, speech and deglutition are affected. The small nucleus of the nerve is thought to control the finer lingual movements of articulation.

CHAPTER VIII.

DISEASES OF THE CRANIAL NERVES.

Diseases of the Olfactory Nerve.

The olfactory nerve is affected clinically by loss of function or anosmia, increased sensitiveness of function or hyperosmia, and perversions of function or parosmia.

Anosmia is far the most common disorder of olfaction.

Etiology.—Its usual cause is disease of the mucous membrane of the nose. Injuries, inflammations, and tumors affecting any part of the course of the nerve, its bulb or central fibres, may also cause it. Unilateral cortical lesions in the uncinate gyrus may lead to partial loss of smell. It will be not entire, because each nerve receives fibres from both hemispheres. Paralysis of the fifth or seventh nerve may indirectly cause some anosmia. Primary degenerative changes and excessive olfactory stimulation cause anosmia. There may also be a congenital absence of the nerves. Anosmia occurs sometimes as a pure neurosis in hysteria or in neurasthenic states.

Diagnosis.—This is made by test odors.

To test the sense of smell, a bottle of oil of cloves or of some familiar non-irritating odor may be used. To detect quantitative disturbance one may use six phials containing oil of cloves, in purity and in watery mixture of 1 to 10, 1 to 100, 1 to 1,000, 1 to 10,000, and 1 to 100,000. Special olfactometers have been devised. The sense of smell for any single odor is lost in about three minutes, but returns after one minute's rest.

Treatment.—For functional anosmia, snuffs containing strychnine gr. $\frac{1}{30}$ and gum acacia ʒ ij. can be used. Weak galvanic and faradic currents are recommended. Usually there is in anosmia a local catarrhal condition of the nose which requires treatment.

Hyperosmia occurs only rarely and then in neurasthenic, hysterical, or insane persons. In the latter it is more often a psychical phenomenon than a peripheral disorder. Hyperosmia can be cultivated, and this is done sometimes by the blind and by those engaged in certain pursuits, such as tea-tasting and wine-tasting.

Hallucinations of smell occur in the insane, as just mentioned,

and a few cases of epilepsy are reported in which the cause was a stench. When all olfactory sensations are disagreeable the condition is called kakosmia.

Parosmia is a not infrequent condition. In it everything smells alike to the patient, and the odor smelled is perhaps a peculiar or offensive one. This condition may be due to local disease, but is often a symptom of hysteria or neurasthenia.

Diseases of the Optic Nerve and Centres.

The optic nerve may be affected by nearly all forms of pathological change. For the neurologist, however, the especially important conditions are inflammations, degenerations, injuries, and functional disorders.

In order to understand the descriptions of these diseases, it is necessary to define some of the symptomatological terms used:

Astheniopia is a condition in which exertion of the eyes is wearisome and painful.

Amblyopia is a partial loss or dimness of vision in which there are no observable lesions.

Amaurosis is a total blindness in which there is no observable lesion.

Hemianopsia is blindness of half the visual field.

Hemiopia is a condition in which there is vision in one-half the visual field.

Scotoma is a name given to blind spots or areas in the visual field.

Inflammation of the nerve, or optic neuritis, may occur as a papillitis or inflammation of the head of the nerve, a neuro-retinitis or descending neuritis, a perineuritis or a retrobulbar neuritis.

Perineuritis is rare. Neuro-retinitis and papillitis are closely associated clinically and pathologically (Noyes), so that practically only two forms of neuritis need be discussed separately.

Papillitis and Neuro-retinitis.—*Etiology.*—The causes in general are tumor and inflammation of the brain, poisons, infections, and auto-toxæmic states. In particular the causes are tumors, abscess of the brain, meningitis, syphilis, myelitis, nephritis, diabetes, infectious fevers, multiple neuritis, lead, and severe hemorrhages. The disease occurs at all ages and in both sexes. In brain tumors it occurs in two-thirds of the cases, and especially often in cerebellar tumors. It occurs in 80 per cent of cases of tubercular meningitis.

Symptoms.—Subjective symptoms are often not present. The vision may remain good for a long time. In other cases there are concentric limitation of the visual field, loss of color sense, and scotomata. For a description of ophthalmoscopic changes, the

reader is referred to special text-books. It is in this condition that "choked disc," which is a papillitis with much serous infiltration, occurs. The changes are less striking in neuro-retinitis. The disease may affect one or both nerves. In brain disease both nerves are usually involved.

Pathology and Pathological Anatomy.—The process is usually subacute or chronic. Congestion, exudation, small hemorrhages, and collections of leucocytes occur. The sheath of the nerve just back of the globe is often distended with a serous exudate. After a time the nerve fibres atrophy, connective tissue proliferates and takes their place, and we have a secondary optic atrophy.

FIG. 59.—NEURO-RETINITIS (JAEGER).

The process is essentially peripheral, but it extends back with lessening intensity into the trunk of the nerve. The purely mechanical theory of neuritis, that it is due to compression, cannot be accepted in the light of modern pathology. It is probable that the neuritis results from an irritating serous fluid which extends down the sheath of the nerve, this sheath being a prolongation of the arachnoid cavity. Mechanical causes lead to constriction, accumulation of the fluid, and compression of the nerve at its periphery, and hence to inflammation. Sometimes, at least, the irritating fluid contains microbes or microbic poisons.

RETROBULBAR NEURITIS.—In this disease the lesion lies chiefly behind the globe. Its causes are especially toxæmia from alcohol and tobacco. It is also due to rheumatic influences, syphilis, lead, and diabetes.

In the acute cases there is usually rather rapid loss of sight, with some pain and tenderness. The ophthalmoscopic changes are relatively slight. In chronic cases, which are usually toxic in origin and due to alcohol or tobacco, or oftenest to both, there are color scotomata or absolute scotomata and amblyopia. There is no pain. The condition is known as tobacco or alcoholic amblyopia.

The *prognosis* of neuritis varies with the cause. If this is removable, as in the toxæmiæ, recovery is the rule. This is a proof that in neuritis the connective tissue is the part chiefly in-

FIG. 60.—PAPILLITIS, "CHOKED DISC," IN A CASE OF CEREBRAL TUMOR.

volved, for a destroyed or atrophied optic nerve does not recover. In many cases, however, atrophy follows the neuritis.

The *treatment* is based on the cause. In acute cases one may use cups, salicylates, the iodides, and mercury; later, the iodides and strychnia. Perfect rest to the eyes should be enjoined.

DEGENERATION OF THE OPTIC NERVE, OR OPTIC ATROPHY.—This condition may be primary or secondary. Secondary atrophy is usually the result of a neuritis. I shall describe here *primary optic atrophy.*

Etiology.—It occurs oftener in men than women (three to one). It occurs as part of locomotor ataxia in less than half the cases; other degenerative diseases of the cord, like multiple sclerosis, may accompany it. Next to the spinal cord, it occurs oftenest with degenerative diseases of the brain, such as multiple sclerosis

and general paresis. Hemorrhages, alcoholism, and lead may be causes. It may occur without known cause.

The *symptoms* are those of gradual decrease of acuity of vision, concentric limitation of the visual field, loss of color sense, dilatation and immobility of the pupil. The sense of sight may remain good for a long time. Ophthalmoscopically, the nerve disc is opaque, grayish, or dirty looking, and often has a cup-shaped or "cupped-disc" appearance. The vessels are smaller and few in number.

The *pathology* and *pathological anatomy* is that of a parenchymatous degeneration with loss of nerve fibres, which are replaced by connective tissue.

The *prognosis* is almost uniformly bad.

The *treatment* is that usually of the cerebral or spinal disease. Mercury, iodides, strychnia, phosphorus, and iron are given. Warm baths and salicylate of soda sometimes have a temporarily good effect. Strychnia in physiological doses gives, however, the best symptomatic results. Electricity is not of any use. Stretching the nerve does no good. Eserine, pilocarpine, and santonin or nitrate of silver may be tried.

The optic nerves and their primary and cortical centres are subjected to various other diseases. So far as these are organic, they will be described in detail under the head of brain diseases. But there are certain symptoms often of functional origin which are best described here. These are: (1) Amblyopia and amaurosis; (2) retinal hyperæsthesia and dysæsthesia; (3) hemianopsia.

Amblyopia and Amaurosis.—*Etiology.*—The causes are injuries and shocks, hysteria, migraine, concussion of the brain, lightning stroke, and severe hæmorrhages. There are also certain toxic causes, chiefly alcohol and tobacco, quinine, and salicylic acid. Other causes are glycosuria, uræmia, and reflex irritations, especially of the trigeminal nerve. Night-blindness and snow-blindness are forms of functional amblyopia.

The *symptoms* are diminution or loss of vision, usually sudden, temporary, and involving both eyes. Amblyopia in hysteria is usually greater in one eye and associated with concentric limitation of the visual field and disturbance of color sense.

Underlying functional amblyopia there may be a peripheral anæsthesia, a psychical or cortical anæsthesia, minute hemorrhages, causing temporary pressure, vascular spasm, and anæmia.

The prognosis is usually good.

The treatment is purely a causal one. In most cases one must examine for drug poison, uræmia, diabetes, migraine, or a hemorrhage.

Retinal or ocular hyperæsthesia is a condition in

which the eye is abnormally sensitive to light. It may be due to exposure to extreme light or to seclusion in a dark room. The neurologist sees it oftenest as a symptom of hysteria (*vide* Hysteria) and perhaps in hypnotic states. It occurs in mydriasis and albinism. It is not to be confounded with photophobia due to irritation of the conjunctiva.

Nyctalopia, or the sense of seeing better in a dim light, is a form of the disease.

Hemianopsia or half-sightedness may be due to a functional or organic disorder of the nerve or its centres. It is a symptom of many lesions and conditions and can only be described generally here.

Etiology.—Its principal functional causes are migraine, lithæmia, gout. Its organic causes are tumors, inflammations, soften-

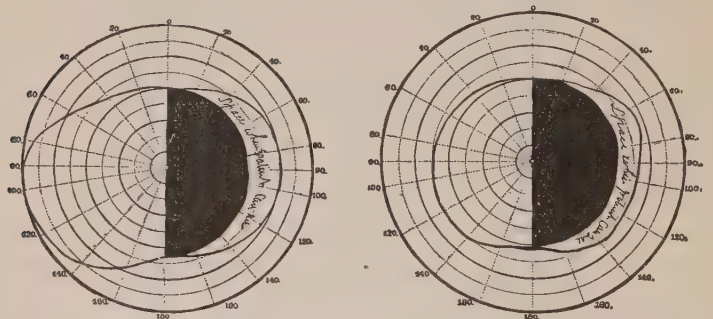


Fig. 61.—Showing Lateral Homonymous Hemianopsia. This is not quite complete, there being a space on the blind side where the patient can see (Noyes).

ings, or hemorrhages involving part of the optic nerve or its central connections.

Symptoms.—Various descriptive terms are used to indicate the character of the hemianopsia. In lateral hemianopsia a vertical half of the field is involved. In lateral homonymous hemianopsia there is half-blindness on the left or right side of each eye, as the case may be (Fig. 61). In temporal hemianopsia the outer half of each eye, and in nasal the inner halves, are involved. The upper or lower segments or irregular segments of the visual field may be involved.

These various forms of hemianopsia depend upon the location of the lesion which cuts into and destroys the optic fibres in their course from the eye to the visual centre in the occipital cortex. The mechanism will be understood when it is remembered that each occipital lobe is supplied by nerve fibres from one-half of the retina of each eye. A cut shows this better than any descrip-

tion (Fig. 49). In nasal hemianopsia the lesion must be at Ch, in front of the chiasm. In temporal hemianopsia it is at C.M., just back of the chiasm. In lateral hemianopsia the lesion must lie farther back than the chiasm, in the tract, the primary centres, the optic radiations, or occipital lobes.

In hemianopsia from disease of the nerve as far back as and including the primary centres in the optic thalamus and corpora quadrigemina, there is a loss of light-reflex when a ray of light is thrown upon the blind side of the retina, but the pupil still contracts when light is thrown on the sensitive side of the retina. This phenomenon is called "Wernicke's hemiopic pupillary reaction." If in hemianopsia the light-reflex is preserved, the lesion is back of the primary centres and involves the optic radiations or cortex.

A test for the condition of hemianopsia in its early stage, and one that is useful in stupid or partially comatose patients, is the following: When the finger is suddenly brought in front of the eye on the sound side, there is a wink; if brought in front from the blind side, the orbicularis does not contract.

Hemianopsia is almost always the sign of organic disease. It is not found in hysteria, but does occur in migraine and lithæmia. It is best made out and recorded by means of the perimeter.

Its course and treatment depend upon the cause.

Diseases of the Motor Nerves of the Eye.

The common affections of the ocular muscles are:

1. Paralyses or ophthalmoplegias, which may be acute, chronic, or progressive. 2. Pareses or amyosthenic states, called ordinarily muscular asthenopias. 3. Spasms, such as strabismus, nystagmus, and blephorospasm.

There are many special terms which are used to indicate the peculiar effects of various paralyses and spasms of the ocular muscles and nerves, and some of these I will define here:

Erroneous projection is a condition in which the patient is unable to judge exactly of the relation of external objects to the body. For this relation is determined by the movements of the ocular muscles, and these being weak, wrong sensations are conveyed to the brain. Vertigo may result from this disturbance of muscular sensation.

Diplopia or double vision is a condition due to the erroneous sensation resulting from eye-muscle palsy, and to the fact that the images of the object fall upon non-corresponding retinal fields. Diplopia is simple or homonymous when the false image is seen on the same side as the affected eye. When a red glass is placed over this eye two images are seen, the red one being on the side

of the eye involved. Diplopia is heteronymous or crossed when the false image is on the opposite side to the sound eye.

Conjugate deviation of the eyes is a condition in which both eyes turn strongly to one or the other side. It may be paralytic or spasmodic. The mechanism is a complicated one and not perfectly understood. In general, destructive lesions of the brain cause a paralytic deviation toward the side of the lesion. Similar lesions in the pons cause a deviation away from the side of the lesion. The palsy involves the sixth cranial nerve and the branch to the internal rectus from the third. The sixth nerve nucleus is the dominant one, and impulses from the brain go to it first (see Fig. 51).

Returning now to the diseases of the motor nerves of the eye, it is found that the paralyses occur in various ways, which may be best grouped as follows:

Ophthalmoplegias:

1. Paralyses of the third nerve. } Acute.
2. Paralyses of the fourth and sixth nerves. } Chronic.
3. Progressive paralysis of all or part of these nerves.

The Ophthalmoplegias.

I. Paralysis of the Oculo-Motorius or Third Nerve. —*Etiology.*—The commonest causes are exposure to cold and syphilis. Other causes are basal meningitis, intracranial tumors, injuries, compression from orbital tumors, the diphtheritic poison, and excessive exposure to light; excessive use of tobacco, and alcohol, morphine, or other poisons may be a cause. Temporary palsy sometimes occurs in migraine, or it may take the place of an attack of migraine. A palsy of some of the muscles supplied by the third is sometimes caused by cerebral lesions involving the inferior parietal lobule. Partial palsies also occur in locomotor ataxia and in certain primary muscular atrophies. The common causes, however, are, as stated, rheumatic influences and syphilis.

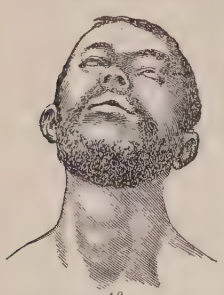


Fig. 62.—Double Ptosis.

There occurs, in rare cases, an acute inflammatory degeneration of the nuclei of the ocular muscle nerves similar to acute anterior polio-myelitis. This condition has been called "polio-encephalitis superior."

Symptoms.—When all the muscles supplied by the third nerve are paralyzed, there is dropping of the lid (ptosis); the eye can only be moved outward and downward and inward; there is there-

fore divergent strabismus and double vision (diplopia). The pupil is somewhat dilated and does not contract to light, owing to paralysis of the constrictors of the iris; and there is loss of power of accommodation, so that the patient cannot read print close to him.

The patient suffers under annoyance from the lid drop and the double vision, and there is sometimes vertigo and photophobia. Only one nerve is involved at a time as a rule. The various eye muscles supplied by the third are rarely all attacked.

FIG. 63.—SHUTTER FOR TESTING PUPIL-REFLEX. The apparatus admits of using colored glasses for testing hysteria and malingering; also of the use of a Maddox prism for testing the eye muscles.

The levator may escape almost entirely the ciliary muscle and iris may also be but slightly involved; but these latter muscles are never involved alone in ordinary types of the disease.

The affection usually runs a subacute course, lasting but a few weeks. Functional palsies last but a few days; syphilitic palsies are usually temporary (one to three weeks), but may relapse or become extremely obstinate. Periodical palsies occur every year or six months or even oftener; they last a few days or weeks and are accompanied at first with some pain. They continue to recur for years.

In diphtheritic eye palsies the first three or four nuclei of the series making up the origin of the third nerve are oftenest affected, causing paralysis of accommodation, paralysis of the iris and of the internal rectus. the three muscles concerned in accommodating the eye to near objects (Fig. 62).

Pathology.—In the rheumatic palsies there is a low grade of peripheral neuritis, and the same is true of most diphtheritic and other palsies of infectious origin. In syphilitic palsies there is usually a specific basilar meningitis involving the nerve roots. The meningitis may be slight or may amount to gummatous deposit. In functional and some periodical palsies there is a vasomotor disturbance causing congestion or anæmia or perhaps simply inhibition of the nuclear centres. Some periodical palsies have been found to be due to small tumors involving the nerve root. In rare cases there is primary muscular atrophy of the eyeball nerves, or primary degeneration of the nuclear centres, or a cerebral lesion of the inferior parietal lobule.*

Diagnosis.—One must first determine how extensively the muscles supplied by the third are involved.

If only the levator palpebræ, there is simply falling of the lid. If the eyeball muscles are involved, we get the following symptoms. They are:

Limitation of movement of the globe.

Strabismus and secondary deviation.

Erroneous projection.

Double vision or diplopia, which is either simple or crossed.

Paralysis of the iris or iridoplegia and of the ciliary muscle or cycloplegia.

Concentric limitation of the visual field.

All these points must be tested, but the detailed knowledge of them is best gained by consulting ophthalmological works.

The extent of involvement of eye muscles can generally be sufficiently tested by making the patient move the affected eye in various directions, and by testing for accommodation and for the pupillary reaction to light.

Paralysis of the iris, or iridoplegia, and of the ciliary muscle—cycloplegia.

The motor fibres of the third nerve to the iris supply the sphincter, and when paralyzed there is dilatation and immobility of the pupil, a condition known as *mydriasis.*

Fibres from the same nucleus innervate the ciliary muscle, and the iris and this muscle are usually paralyzed together. Paralysis of the ciliary muscle is called *cycloplegia.* In this latter condition there is loss of power of accommodation. Iridoplegia and

* Some diphtheritic palsies are probably nuclear.

cycloplegia are usually due to local disease of the eye or to the use of mydriatic drugs. Occasionally they are observed after diphtheria or in multiple sclerosis.

Paralysis of the levator palpebræ, causing *ptosis*, is sometimes seen alone, but usually other branches of the third nerve are involved.

A functional palsy of the lids sometimes occurs in anæmic and nervous people at the time of waking. It is a temporary *morning* or *waking ptosis*.

Paralysis of the sympathetic fibres of the eye causes contraction of the pupil (myosis) from the unopposed action of the third nerve. There is also a slight prominence of the eye and slight ptosis from an involvement of the nerves that supply Müller's muscle. The pupil does not dilate when the skin of the cheek or neck is irritated. This is a condition known as loss of skin-reflex. In locomotor ataxia there is often a rigidity of the constricting fibres of the iris, while the ciliary muscle continues to act. The pupil is then small and does not respond to light, while it does respond to accommodation. This is known as the *Argyll-Robertson pupil*.

II. Paralysis of the Fourth Nerve.—This is a rare affection and not always easily detected. The causes are much the same as those of palsy of the third nerve.

The symptoms are slight convergent strabismus when the eye is moved downward and diplopia on looking down. There is defect in the movements of the eye downward and outward.

Paralysis of the sixth nerve (abducens) is the most frequent of eye palsies, and occurs especially often in syphilis and in locomotor ataxia. It causes convergent strabismus and double vision.

III. Progressive Ophthalmoplegia.—Besides the palsies already described, there occur certain forms which have a peculiar origin and course. They begin slowly, as a rule, and steadily progress. In some cases only do they reach a certain stage and then remain chronic. The term "progressive" applies fairly well to them. They often affect the third, fourth, and sixth nerves together. In accordance with the muscles invaded, these palsies are called *external*, *internal*, *partial*, and *total*. Thus if those branches of the third nerve supplying the iris and ciliary muscle are involved alone, it is ophthalmoplegia interna; if the other branches are involved, it is called ophthalmoplegia externa.

Definition.—Progressive ophthalmoplegia is a degenerative disease of the nuclei of the motor nerves of the eye. It is in most cases the same disorder as of bulbar paralysis and progressive muscular atrophy. It is sometimes called polio-encephalitis superior.

Etiology.—It develops between the ages of fifteen and forty, but may occur later. The sexes are equally affected. Lead, diphtheria, traumatism, syphilis, appear sometimes to be the cause. It may complicate locomotor ataxia; more often it forms part of progressive muscular atrophy.

The *symptoms* are often not noticed until the disease is far advanced. The vision is not disordered, and there is only a gradual limitation of mobility of the eyeball. A slight drooping of the lids, causing a sleepy look, or a slight squint, usually divergent, is noticed. Then upon examination it is found that the eye cannot follow the finger, except to a slight extent. The iris reacts to accommodation and light usually. Double vision may be present. Usually the patient accustoms himself to monocular vision. The disease lasts a long time, and it may become stationary. If complicated with progressive muscular atrophy, however, the course is relatively rapid, death occurring from the latter disease in two or three years.

Pathological Anatomy.—In all progressive cases there is a degenerative atrophy of the nuclear cells. In a few rare cases no lesion has been found, and in a few stationary cases the anatomical change is that of neuritis.

The *treatment* is that for the disease which it complicates or the condition which causes it. That is to say, it is the treatment for locomotor ataxia, progressive muscular atrophy, syphilis, or lead-poisoning. Iodide of potassium, strychnia, arsenic, nitrate of silver, and phosphorus may be given. Electricity is of very doubtful value, and only the galvanic current would be indicated. General tonic measures and rest to the eyes should be employed.

Muscular Asthenopia and Muscular Insufficiencies.—This is a term employed to indicate a lack of equilibrium of the muscles of the eye, as a result of which the visual axes cannot be kept parallel without an effort. This effort is often unconscious, and shows itself only by a ready tiring of the eye on attempting to read, or by the production of headaches and cerebral paræsthesiæ.

Examination of the eye by means of prisms reveals the special character of the trouble.

When the eye muscles act normally the condition is called one of *orthophoria.*

When some of the muscles are weak it is called *heterophoria.* There are various forms of heterophoria, viz.: esophoria, a tending of the visual lines inward, from weakness of the externi; exophoria, a tending of visual lines outward; hyperphoria, a tending of visual line of one eye above its fellow.

The condition is tested in various ways. The simplest is this:

Refractive errors having been corrected, a series of prisms is placed over the eye at first with the base inward while the patient looks at a candle twenty feet distant. The prisms are increased in strength until the patient can no longer coalesce the images. The degree of prism is noted, and this indicates the strength of abduction or of the externi.

The same process is gone through with for the interni, the base of the prism being out.

The externi should overcome a prism of about 8°, the interni one of 23° to 25° or more. There are great individual variations, and there is also considerable variation in individuals.

The above tests measure the amount of abduction and adduction.

To test the presence of heterophoria, the writer uses the Maddox double prism held in a frame. The line where the bases of the two prisms unite is brought directly over one eye, and is held there in a perfectly horizontal position while the patient looks at a candle twenty feet away. A red glass is at the same time held over the other eye. With the eye covered by the double prism the patient sees double, one flame being above the other, with the other eye he sees a red flame lying just between the two white ones. If the red flame is directly in a vertical line, there is orthophoria, but if it lies to one side or the other, there is exophoria or esophoria according as the red flame was on the opposite side to the eye covered with red glass or on the same side. If heterophoria is found, prisms are placed over the eye until the three lights are in a vertical line. The number of the prism required to correct the heterophoria indicates its extent in degrees.

By changing the double prism so that its common base line is vertical, the test for hyperphoria can be made.*

Muscular asthenopia is said to cause a disturbance of vision, vertigo, migraine, cerebral paræsthesia, and pains in the head, more particularly in the occipital and cervical region. It is believed to be a possible factor in producing choreic twitchings in the face. In neurasthenic persons it may cause a wider range of nervous symptoms. It is said to be an essential factor in causing epilepsy, chorea, and hysteria. The author cannot accept this latter view, and believes that the importance of muscular asthenopia in causing general nervous symptoms is not great. Much of it, if not all, may be relieved by correcting refractive errors and by helping the general health of the patient.

The treatment of it, after all myopia, or hypermetropia, or astigmatism, if present, is relieved, consists in the systematic use of prisms for training the muscles, the wearing of proper glasses,

*More elaborate and exact methods have been devised by Dr. Stevens and are employed by many ophthalmologists.

and graduated or complete tenotomies according to the method of Stevens.

Spasmodic Diseases of the Ocular Muscles.

These are: (1) Conjugate deviation from spasm; (2) irregular and associated spasms from convulsive and irritative brain disorder; (3) nystagmus.

Etiology.—Spasmodic conjugate deviation occurs from an irritating lesion of the ocular nuclei or of the brain in its cortical motor areas and tracts.

Irregular spasmodic movements occur in meningitis, hydrocephalus, and in lesions involving the semi-circular canals. Peculiar associated spasms occur in hysterical attacks.

Various spasmodic movements and contractions of individual eye muscles occur from ocular disease, errors of refraction, muscular weakness and paralysis of certain eye muscles.

Rhythmical spasm or *nystagmus* occurs as the result of hereditary visual weaknesses and refractive errors of various kinds, in albinoes, and in chronic hydrocephalus. It is found usually in neurotic cases associated with ocular defects, in epilepsy, chorea, hysteria, neurasthenia, and insanity. It occurs in certain degenerative nervous disorders such as disseminated sclerosis, hereditary ataxia, tumors, especially of the cerebellum, and other focal lesions, and in meningitis. It occurs in miners, and is called miners' nystagmus. It may be a reflex symptom from a remote irritation.

In nystagmus the oscillation of the eyeballs is usually lateral. It may be brought out when slight in degree by causing the patient to look steadily to one side.

Vertical and a kind of rotating nystagmus sometimes occur, and are due to much the same causes as those of lateral nystagmus.

Spasm of the levator palpebræ is sometimes seen, and is usually tonic.

DISEASES OF THE FIFTH CRANIAL NERVE—THE TRIGEMINUS.

The common affections of this nerve are headache, migraine, tic douloureux, and various neuralgias. The rarer diseases are anæsthesia, reflex cough and asthma, masticatory paralysis, trismus, trophic and secretory and vasomotor disorders.

Many of the disorders referred to the fifth nerve are in reality only symptoms of some general neurosis, constitutional disturbance, or of some remote local disease. As a matter of convenience, however, they may be treated here.

Headache (Cephalalgia).

Headache is the name given to attacks of diffuse pain affecting different parts of the head and not confined to the tract of a particular nerve. It usually comes on in paroxysms at various intervals, but may be continuous.

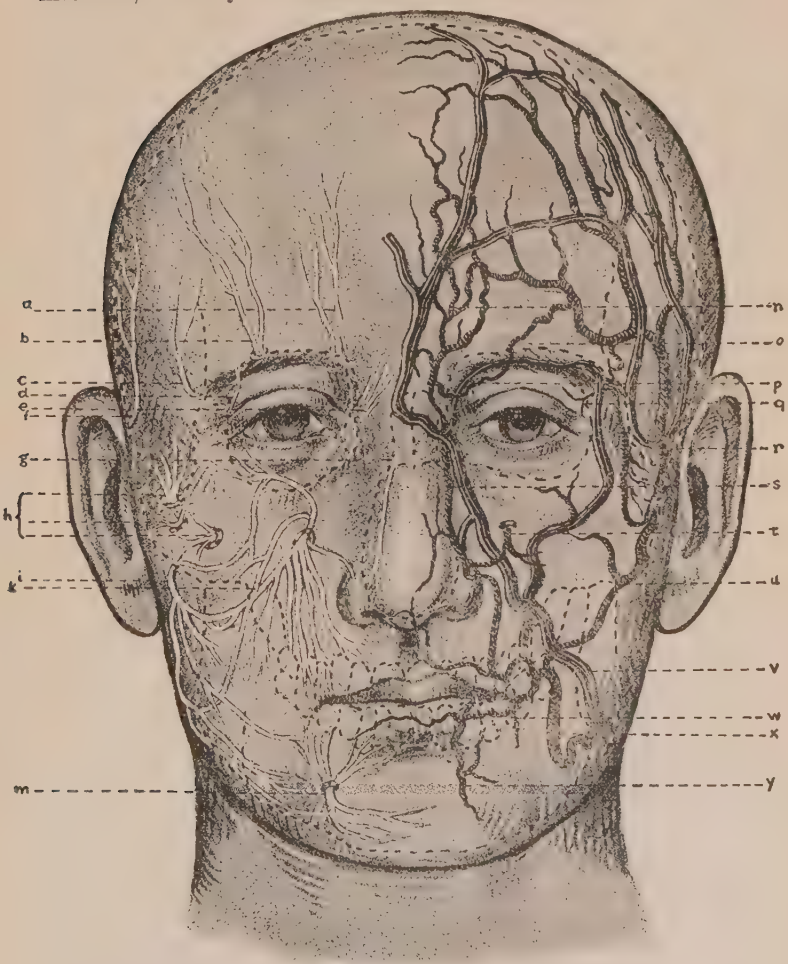


Fig. 64.—Showing the Motor and Sensory Nerves and the Arteries and Veins of the Face (Merkel). *a*, Frontal nerve; *b*, supra-orb.; *c*, temporal; *d*, lacrymal; *e*, supra- and infra-trochlear; *f*, auriculo temp.; *g*, nasal; *h*, malar; *i*, seventh nerve; *k*, infra-orbital; *m*, mental. The veins have longitudinal, the arteries oblique striations. The nerve *i* is the facial; all the other branches are trigeminal.

Etiology.—Headache is the most common of nervous symptoms. Ten to fifteen per cent of school children, twenty-five per cent of men, and over fifty per cent of women are subject to it more or less.

The headache ages are from ten to twenty-five and thirty-five to forty-five; most cases occur between the ages of eight and twenty-five, especially in females. The number of headaches increases gradually from the period five to ten years up to the period fifteen to twenty, then falls till the thirty-fifth year, and rises again till about the age of forty. Early childhood and declining age are practically exempt from chronic functional headaches. Women suffer from it more than men in the proportion of about three to one. It is more frequent in city populations and among the wealthier classes. Headaches are more common in the spring and fall and in temperate climates. Headaches may be classed, in accordance with their causes, as follows:

1. Hæmic causes, in which impoverished or disordered blood is brought to the brain, as in (*a*) anæmia and congestion; (*b*) diathetic states: gout, rheumatism, diabetes, uræmia; (*c*) infections: malaria, fevers.

2. Toxic causes: lead, alcohol, tobacco, etc.

3. Neuropathic states: epilepsy, neurasthenia, hysteria, neuritis.

4. Reflex causes ocular, naso-pharyngeal, auditory, dyspeptic, sexual.

5. Organic disease, including arterio-sclerosis, syphilis, tumors, meningitis, and diseases of the cranial bones. The anæmic, dyspeptic, ocular, and neurasthenic are the common forms of chronic and recurrent headache.

Symptomatology.—Headaches may be classed in accordance with their location and the character of the pain. We have accordingly: 1. frontal headaches; 2, occipital headaches, 3. parietal and temporal headaches; 4, vertical headaches; 5, diffuse headaches and various combinations of the above.

The most common form of headache is the frontal, next the fronto-occipital or diffuse, next the vertical, and then the occipital.

The kind of pain differs with different persons and with different causes. We have: 1, pulsating, throbbing headache, 2, dull, heavy headache; 3, constrictive, squeezing, pressing headache; 4, hot, burning, sore sensations; 5, sharp, boring pains.

The first form characterizes headaches with vasomotor disturbances, and usually indicates migraine.

The second is of a toxic or dyspeptic type.

The third is found in the neurotic and neurasthenic.

The fourth in rheumatic and anæmic cases.

The fifth in hysterical, neurotic, and epileptic cases.

The accompanying diagram shows some of the relations of localized pain to the cause. A large experience both in my own practice and in that of others shows it to be approximately correct.

Headaches may continue for a day or may last for weeks and months. Some persons have headaches only when constipated or bilious, or when they have an attack of indigestion. Others suffer from a little pain nearly all the time, exacerbations occurring at various periods. Neurasthenic and ocular headaches are generally of this type. When headaches are persistent, examination should be made of the eyes, of the nose and sinuses; the patient should be questioned as to syphilis, the continuous use of tobacco, and chronic dyspepsia. The possibility of brain tumor, of pachy-

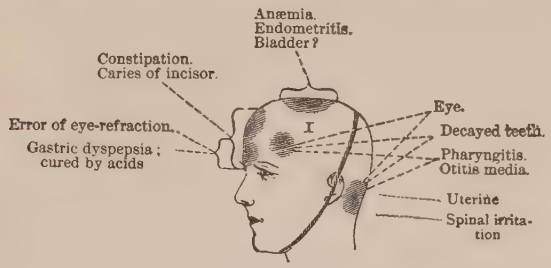


Fig. 65.—Showing the Location of the Pain in Headaches from Different Causes.

meningitis from blows, or sunstroke or chronic alcoholism should be considered.

The persistent headaches not relieved by ordinary treatment are due to eye trouble, anæmia, neurasthenia or spinal irritation, syphilis or pachy-meningitis. Eye strain may cause true migraine or ordinary headache. Eye-strain headache is usually asociated with some weakness of eyesight and pains and discomfort about the globe, besides severer pains at times in the brow or occiput. The cause of the eye strain is usually astigmatism and hypermetropia. Occasionally it is due to weakness or lack of balance of the eye muscles.

Symptoms Associated with Headache.—The symptoms oftenest associated with chronic and recurrent headaches are vertigo, somnolence, sensations of heat and pressure (cerebral paræsthesiæ), and nausea. Vertigo goes oftenest with headaches of dyspeptic origin; some of the so-called bilious headaches of early life develop later into attacks of vertigo; this symptom often occurs with frontal headaches. Somnolence occurs oftenest with anæmic and malarial headaches; it may develop also with syph-

ilitic head pains. Nausea I have found oftenest with occipital headaches.

Pathology.—Headaches are to be distinguished from neuralgias and from a special and common form of head pain known as migraine.

Headaches are diffused pains caused, as a rule, by irritations located in or referred to the peripheral ends of the fifth nerve. Their seat is usually within the skull.

Neuralgias, on the other hand, are caused by irritations of the trunks of these nerves. The pains are local and confined to the single branches of the nerve.

Migraine is a periodical neurosis in which there is a discharge of nerve force, not only affecting the trigeminus, but often other cranial nerves as well as sympathetic fibres.

It is a general disease of which the headache is only one symptom.

The nerves of the dura mater are those most involved. Headaches, when occipital, involve the sensory fibres of the vagus and the upper four cervical nerves.

There is no anatomical change in the nerves except in organic headaches. But in many cases the membranes of the brain and their sensory nerves are congested or anæmic.

Diagnosis.—No symptom requires more careful investigation as to its cause than that of headache. The diagnosis is always to be made, not of this symptom, but of its cause. Most of the foregoing description accordingly refers to etiology.

It is important, however, to decide whether the case is one of migraine, or neuralgia, or of headache.

Headache is usually diffuse and bilateral. It is more or less persistent. Migraine comes on paroxysmally, lasts a short time, and then leaves the patient feeling perfectly well or even better than ever. Migraine is often accompanied with nausea, flashes of light, strong pulsations of the head, vertigo, pallor or, more rarely, congestion of the face. Neuralgic pains are sharp and shooting, they run along the tract of the nerve, and often are associated with suffusion of the eye and œdema. Tender points are felt.

Treatment.—The constitutional treatment is based upon the etiology. Regulation of diet and digestion, securing a regular movement of the bowels, attention to ocular troubles, abstention from tobacco and alcohol and overwork are the important points requiring attention.

The symptomatic treatment consists in giving antipyrine, antifebrin, phenacetin, salicylate of soda, caffeine, muriate of ammonia, and sometimes morphine or codeia, and bromide of ammonia.

Antipyrine can be given in doses of gr. v. every twenty minutes

till three or four doses are taken. Phenacetin often needs to be given in large doses of ten or even twenty grains. Antifebrin is less trustworthy and must be given in small doses. Exalgin is not a very good or safe remedy. It may be tried in doses of gr. iij. to gr. v. Muriate of ammonia is an excellent remedy given in very large doses, ʒ ss. to ʒ i., well diluted. Menthol in doses of gr. v. to gr. x. in hot water sometimes stops headaches. In headaches from anæmia, caffeine and ammonium muriate are best. In headache from nervous exhaustion, similar stimulating anodynes are usually most efficacious. Combinations of caffein citrate and salicylate of soda or benzoate of soda are often better than the single drug. Caffeine in any case must be given in larger doses than is ordinarily done, *i.e.*, gr. iv. or v. Local applications of a twenty-per-cent solution of menthol, the ice-bag, cloths wrung out in hot water, or a piece of sheet lint soaked in chloroform liniment two parts and tincture of aconite one part are efficacious measures. A cathartic, rest in a darkened room, light diet—all these are measures which many patients themselves learn to adopt.

Finally, in headaches from organic disease we have often to resort to iodide of potassium, mercury, and the use of some preparation of opium.

As will be seen, each case of headache requires special treatment and a certain amount of experimentation in order to learn the idiosyncrasy of the patient.

Migraine, Sick-Headache, Hemicrania.—Migraine is a constitutional neurosis characterized by periodical attacks of pain chiefly in the course of the fifth nerve. The pain is often associated with nausea or vomiting, mental depression, vasomotor disturbances such as flushing or pallor of the face, by flashes of light, vertigo, tinnitus aurium, and in rare cases by partial paralysis of one oculo-motor nerve.

It will thus be seen that migraine is more than ordinary headache and unlike an ordinary neuralgia.

Etiology.—The disease is very common in civilized countries and is frequent in America. It occurs oftenest in women in the proportion of about three to one, and it begins in most cases at or a little before the age of puberty. It may begin as early as the fifth or even the second year. It occurs in neurotic families, and there is very often a history of direct inheritance. Other neuralgic troubles, epilepsy, and gout may be found in the family history.

The attacks occur oftenest in the winter in our climate.

The cases that begin in childhood and early life are sometimes started by overwork at school, but usually no especial cause can be found. When they begin after maturity, a history of

excesses in work, injury, shock, or exhausting disease is found. Migrainous patients usually have some refractive disorder of the eye or a weakness of eye muscles, and this condition is one factor in bringing on or keeping up the headaches.

Symptoms.—The patient for several days may feel a sense of malaise and depression; usually, however, the prodromal stage lasts only a few hours or a day. The attack often comes on in the morning and gradually increases in frequency until the victim has to give up work and lie down. The pain starts in one side of the head, usually in the forehead, but often in the occiput. It increases and finally may involve the whole head. The pain is of a tense, throbbing, blinding character, increased by jars, light, and noises. It is accompanied by dimness of vision, often by flashes of light or dark or light spots variously colored floating before the eyes. Restriction of the visual field, sometimes in the form of hemianopsia, may occur. Vertigo, tinnitus aurium, confusion of ideas, feeling of stupor, disturbances of memory, are not uncommon; nausea and even vomiting are the rule. The vomited matter is at first chiefly mucus, but it may later get yellow and bitter from the presence of bile. Hence the term "bilious headache," which is an improper one, because the bile is only the result of retrostaltic action from the vomiting. Migraine is not the result of gastric or liver disorder.

The patient's face usually is pale and gives the evidence of acute suffering. The flushed face is very rare; the distinction between angiospastic or pallid migraine and angioparalytic or congestive migraine is not of clinical value.

The pulse is small and hard and may be lessened in rapidity.

The temperature often rises in children.

The attack lasts from six to twelve or twenty-four hours, occasionally even two or three days. As the intensity of the pain lessens the patient sinks to sleep, and awakens next morning feeling refreshed and better than before the attack.

The attacks occur at varying periods, fortnightly or monthly, and even weekly. In women they often occur during menstruation. Some women are entirely free from them during pregnancy. At about the time of the menopause in women, and at about the same time of life in men, the disease lessens in severity and as a rule disappears. Some form of neuralgia or some neurosis in rare cases takes its place.

Complicating Symptoms.—Partial oculo-motor paralysis, temporary aphasia, slight hemiplegia, hemi-numbness, peculiar odors or tastes, convulsive movements of the body, almost resembling epileptic attacks, occur.

Cases presenting these symptoms are rare. When they occur in one case, however, they always occur in each attack unless it is modified by treatment.

Vicarious Attacks.—Migraine is sometimes associated with epilepsy, locomotor ataxia, or insanity; that is to say, persons in early life have had migraine and later developed the diseases mentioned. The relation between these diseases, however, is not so close as some writers would lead us to infer. The attack of migraine can be sometimes replaced by an attack of gout, or visual disorders, or other sensory symptoms, or even by an oculomotor paralysis. Sometimes, instead of a fully developed attack the patient has a sense of mental depression, with confusion of ideas. Cases have been reported in which acute mania took the place of the headache.

Types.—Writers have described the angioparalytic and angiospastic types; also the typical, subtypical, and supratypical; and finally the ordinary type and the ophthalmic type. Practically we find two classes:

(1) The typical, associated with visual disorders and having most of the symptoms described above.

(2) The irregular or mixed type in which with many symptoms of ordinary migraine there is a history of rheumatic influences, and often of anæmia or dyspepsia. These are cases of a true migrainous affection complicated with some form of symptomatic headache, such as has been already described. The mixed or irregular migraines are important to recognize, for they call for special treatment. They occur almost altogether in women; the attacks are often associated with weather changes and may be of a neuralgic character. Many women have their sick-headaches and their neuralgic headaches, so called, and have to distinguish between them. In mixed attacks the face is usually also pale, the eyes are not suffused, nor is there any visual or aural disturbance, as in migraine. Sometimes the pain remains in the occipital nerves, and I have met with one patient who localizes her pain there entirely, and who always vomits during the attack.

Pathology.—The seat of the disease is chiefly in the intracranial branches of the fifth nerve and of the pneumogastric; the upper cervical nerves, however, are often involved. There are no morbid anatomical changes known.

The most plausible theory of the disease is that it is a fulgurating neurosis, in which there are periodical discharges of nerve force, or nerve storms. The seat of the discharge is perhaps in the cerebral cortex, but more likely in the primary sensory centres, *i.e.*, the root ganglia of the fifth and vagus nerves. The disease is certainly not in the sympathetic system, as was once taught.

Diagnosis.—The diagnosis is based upon the hereditary history, the periodicity and seat of the attacks, the nausea, the complicating visual and other sensory symptoms. It should not be

forgotten that the same patient may have migraine and other neuralgias, or may have also an organic brain or renal disease.

Treatment.—Prophylaxis.—Children of families in which this neurosis exists should be carefully watched during the ages between five and twenty. The eyes and nose should be examined. They should not be subjected to excessive mental or visual strain, and if attacks develop they should be promptly treated. The application of glasses should be considered, but not hastily adopted.

As regards *constitutional treatment*, the best measures for curing a case of migraine consist in correcting any visual or nasal defect, such diet and exercise as secure tone to the nerves, and the continuous use internally of bromide of potassium, nitroglycerin, cannabis indica, or arsenic.

Of the above drugs, cannabis indica, either with or without arsenic, is the most trustworthy, while the bromides are the least. The hemp should be given in large doses and for a long time, gr. ½ to gr. i. ter in die. Much stress is laid by some upon ocular muscular insufficiencies, and I believe that such conditions should be remedied, but place little confidence in them alone. On the other hand, the correction of small or large degrees of astigmatism and hypermetropia sometimes produces surprisingly good results. The reported cure of numerous cases of migraine by treatment of nasal hypertrophies and catarrh should not excite too much confidence in such measures. In fact, since migraine is a constitutional neurosis, one cannot expect permanent results from removing reflex irritants alone. The daily use of a strong galvanic current four to eight milliamperes (eight to ten cells) for ten minutes is useful.

For *the relief of the attack* the following drugs may be given: Salicylate of soda, gr. xx.; caffeine, gr. ij. to v., with benzoate of soda, gr. x.; antipyrine, gr. v. q. ½ h.; phenacetin, gr. x. to xv. or more; powdered guarana, gr. xx.; menthol, gr. v. to x. in hot water; muriate of ammonia, gr. xxx. to ʒ i.; bromide of potassium, gr. xx. Antipyrine and phenacetin are the most certain of the above drugs, but they lose their effect after a time, as do almost all the other drugs, and finally patients give up treatment or resort to codeia or morphine. Chloral and a hot foot-bath break up attacks sometimes.

Locally galvanic currents are sometimes helpful, and so are static sparks. Hot applications and pencilling with menthol give relief to some. Quiet and rest are spontaneously resorted to.

Neuralgias of the Trigeminus.—The trigeminal nerve is subject to two types of neuralgia, viz.:

1. The symptomatic form.
2. Tic douloureux.

1. The *symptomatic neuralgias* are by far the most frequent.

They are called supra-orbital, infra-orbital or supra-maxillary, infra-maxillary or dental, and mixed forms. The most common type is the supra-orbital; next, the mixed form.

Etiology.—The female sex is oftenest affected; most cases are seen in the first half of life; most attacks occur in the winter and spring. The left side is oftener affected. The second and third branches of the fifth nerve are most susceptible to rheumatic influences, the first branch to malarial and septic poisons. Dental disorders naturally are frequent factors in neuralgia of the second and third branches. Anæmia, exposure, child-bearing, and other depressing influences are factors in causing these neuralgias. Ocular and nasal disease may cause pain in the supra-orbital nerve. Gout, diabetes, syphilis, malaria, hysteria, epilepsy, trauma, rheumatism, all may be factors in causing trigeminal neuralgias.

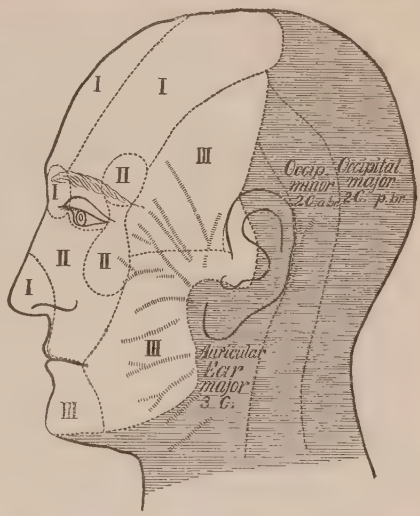


FIG. 66.—SHOWING THE DISTRIBUTION OF THE SENSORY NERVES OF THE FACE. *I, II, III,* First, second, and third branches of the fifth. The shaded part is supplied by the cervical nerves; 2 *C. a. br.*, second cervical anterior branch; 2 *C. p. br.*, second cervical posterior branch.

Symptoms.—The pains of trigeminal neuralgia are sharp and intense, with exacerbations and remissions. The pain often lasts for days without entirely ceasing. It then goes away and may not return for a long time. The characteristic of neuralgia is that it stays till its cause is removed. In supra-orbital neuralgia there is sometimes great œdema of the lids and the parts about, together with suffusion of the eyes. There are tender points over the course of the nerves. The common points in supra-orbital neuralgia are supra-orbital, palpebral, and nasal; in infra-orbital neuralgia, nasal, malar, and gingival; in infra-maxillary neuralgia, inferior dental and temporal. In the mixed forms we find various combinations of the above. There is often also a tender point over the parietal eminence and vertex.

The pain may radiate to the ear or occiput or it may be located in the orbit. Dental irritation may also cause an otalgia or a neuralgia in the upper branches of the fifth.

There is sometimes a dilatation of the pupil, and in severe cases a reflex facial spasm occurs.

Tic Douloureux (*Prosopalgia*, *Fothergill's Neuralgia*, *Epileptiform Neuralgia*).—Tic douloureux is a special form of trigeminal neuralgia, unusually severe in its symptoms and obstinate in its course. It ought to be distinguished sharply from the ordinary forms of trigeminal neuralgia. These latter are symptomatic pains almost altogether; while tic douloureux is an idiopathic disorder, or at least is one dependent upon changes in the nerve itself.

Etiology.—It occurs, as a rule, in persons who are over forty, and is seen in the very aged. It is indeed the only neuralgia which old people have. It is brought on by exposure, overwork, and depressing influences; sometimes, perhaps, by local diseases of the teeth and jaws. It occurs in men and women in about equal frequency, in my experience.

Symptoms.—It is characterized by intense, darting pains which usually start in the upper lip and by the side of the nose. From here they radiate through the teeth or into the eye and over the temple, brow, and head. They are confined to one side of the head. During a paroyxsm the face usually flushes, the eyes water, the nose runs, and the patient assumes an expression of the greatest agony. The attack lasts for a few minutes, then becomes somewhat less, but the pain rarely ceases entirely. A breath of cold air, speaking, eating, putting out the tongue—all bring on paroxysms. The pains are worse in winter and often become less or cease during summer. Occasionally they come on for a few months every year, usually during the spring.

Spasmodic movements of the face may be associated with the pain.

Examination rarely reveals any objective trouble, but in a few cases some anæsthesia may be noted.

Pathology.—The disease is usually a degenerative one, and probably is due to irritative and atrophic processes occurring in the nerve. A low grade of neuritis, perhaps from alveolar disease, has been found sometimes, but as a rule the nerve does not appear much changed. The arteries supplying the nerve, however, often undergo the changes of endarteritis, their calibre is much lessened, and the nerve cannot get its proper supply of blood. Thus an obliterative arteritis underlies some cases of the disease.

Treatment.—In cases which occur in old people, the use of nitroglycerin given in doses of gr. $\frac{1}{100}$ q. 2 h. sometimes has a happy effect. The best single remedy, however, is undoubtedly crystalline aconitia given in doses of gr. $\frac{1}{200}$ until its physiological effect is obtained. After this come injections of cocaine. Be-

sides these measures, galvanism daily, iodide of potassium in large doses, gelsemium, croton chloral, codeine, external applications of menthol, freezing with chloride of methyl, and heat—all may be found useful. The common practice of pulling out all the teeth is almost always unsuccessful, and ought not to be undertaken without specially good reason. Tonics containing iron, phosphorus, quinine, or arsenic are generally helpful, and should always be given after a course of specifics. In younger patients the remedies recommended under the head of Migraine and Headache may be given. Change to a warm, equable climate may be tried; it is not a certain resource.

Finally, surgical interference may be necessary. The removal of the nerve at as deep a point as possible is the only operation to be seriously entertained. This sometimes causes cure, but, as a rule, the pain comes back in six or twelve months. Even such a respite, however, is often gladly seized upon. Removal of the Gasserian ganglion has been attempted with some success. Ligature of the common carotid has been tried also, but of late years the operation has been generally abandoned. Simple drilling out of the infra-orbital nerve with a dental probe made of piano wire has given long relief.

There are numerous methods of operating upon the different branches of the trigeminus. For buccal neuralgia, Zuckerkandl has devised a method. For superior maxillary neuralgia, the method of Carnochan, modified by Abbe, is, in my opinion, the best. Others favor Langenbeck's method. Ullmann, Mikulicz, Obalinski, and many others have devised special methods.

Hartley, of New York, has devised an operation by which he enters the middle fossa through an opening in the temporal bone, thus reaching the root of the nerve.

Trigeminal Paræsthesia.—Sometimes persons suffer from peculiar numbness, thrilling, or formication in the course of the trigeminus.

The sensation may be nearly constant and excessively annoying. It never amounts to actual pain.

It occurs in anæmic, nervous, and hysterical persons. It is to be regarded as an abortive form of neuralgia, and so treated.

Trigeminal Anæsthesia.—This occurs from various pathological lesions in the course of the nerve or in its nuclei.

The most common organic cause is syphilitic disease of the membranes at the base of the brain.

Trigeminal anæsthesia occurs together with anæsthesia of other areas in hysteria and in organic disease of the nerve centres. It is sometimes noted in tic douloureux and facial hemiatrophy.

Herpes, *flushing*, *pallor*, *lachrymation*, *salivation* are all symptoms of disturbance of the trophic, vasomotor, and secretory

fibres running in the trigeminal nerve. They are usually, if pathological, only concomitant symptoms of other diseases.

The diseases of the motor branch of the trigeminus are rare, and generally symptomatic of some more general disorder.

Trismus (lock-jaw) is the only important independent affection of this motor nerve. It is a tonic spasm of the muscles of mastication.

Etiology.—It occurs in infants, usually through infection from the umbilicus. It is then known as trismus nascentium. It forms part of the symptoms of tetanus. It may be symptomatic of brain disease, and forms one of the manifestations of the epileptic fit. There may be a reflex trismus from irritations of the teeth and jaw, and from gastro-intestinal trouble. Neuritis and hysteria are causes of trismus.

Symptoms.—There is, as shown above, a symptomatic, an infectious, a reflex, a neuritic, and a hysterical trismus. In all, the symptoms are very manifest. The jaws are firmly locked and the masseters and temporals stand out. If the disease is unilateral, which is rarely the case, the lower jaw is pushed over toward the sound side.

The *treatment* of both tonic and clonic spasms depends upon the cause. Symptomatically, morphine is to be given, and later the antispasmodics, such as the bromides. In rheumatic cases hot applications and diaphoretics are indicated.

Diseases of the Facial Nerve.

The facial nerve being motor, its diseases are spasmodic and paralytic. The two common types are facial tic and facial palsy, but there are other minor forms.

The sapsmodic disorders are (1) diffuse facial spasm or mimic tic and (2) spasm of single branches, including (*a*) blepharospasm and (*b*) nictitating spasm.

Facial Spasm (Mimic Tic).—This is a disease characterized by intermittent, involuntary twitchings of the facial muscles. It is always chronic and generally unilateral.

Etiology.—It is a disease of middle and later life, and occurs oftener in women; there is usually a neuropathic constitution: it is not hereditary. The exciting causes are anxiety, shock, injury, and exposures. It often has a reflex cause, usually from irritation of some branch of the trigeminus or the cervico-brachial nerves, rarely from eye strain. Organic diseases, such as tumors and softening, affecting the nerve nucleus in the pons or the cerebral centres, cause a symptomatic tic, but not the true disease.

Thus we may have a post-hemiplegic tic or a tic due to cortical lesion and associated with epilepsy.

True facial tic may be caused by irritation or disease of the nerve.

Symptoms.—The disease usually begins slowly and the orbicularis muscle and zygomatici are earliest affected. It rarely goes above the eyebrows, *i.e.*, to the corrugator supercilii and frontalis. The lower branch of the facial is little affected. The spasm is a clonic one, the muscles of the face are affected by a series of lightning-like twitches, with intervals of rest. Sometimes the contraction becomes tonic and lasts several seconds or more. There is no pain. The spasm is increased by emotions, nervous excitement, conversation, exposure to light and cold, and is at its worst when the patient himself is most depressed. It is a very good gauge of the general nervous stability of the patient.

There is no paralysis or atrophy, and there are no secretory or trophic symptoms. The taste fibres are rarely involved, though occasional subjective sensations of taste have been felt.

The electrical irritability is either unchanged or slightly increased.

Associated movements of the eyes, of the jaw muscles and cheek muscles are sometimes seen. Pressure over the motor points of the nerve will sometimes arrest the movements for a time. The disease is a unilateral one. It lasts for years and even for the lifetime.

Pathology.—There is no known anatomical change in idiopathic cases. The disease is allied in character to wry-neck and other chronic tics. It is a motor correlative to the severe neuralgias like tic douloureux. It is sometimes a pure reflex neurosis from ocular or dental irritations.

Diagnosis.—Idiopathic facial spasm is chronic, unilateral, unaccompanied by pain or paralysis. It is distinguished from facial spasms of organic origin by the fact that the latter always have some other symptoms. Thus facial habit-chorea is bilateral spasm from cortical disease is attended by disturbance of consciousness and comes on in paroxysms; the spasm occurring after hemiplegia is usually tonic, and so is hysterical facial spasm.

Prognosis.—The disease is in most cases incurable, especially after it has lasted some time. If a reflex cause exist, the prognosis is better. Life is, however, never endangered by it.

Treatment.—The most important thing is attention to the general health, removal of all depressing influences, rest, and freedom from excitement. Among specific remedies arsenic, the bromides, cannabis indica, gelsemium, conium, hyoscyamus, strychnia, codeine, and morphine are recommended. Morphine is

useful, but must be tried carefully and in small doses. Conium lessens the spasm, but this drug has to be given in large doses and is not entirely free from danger. Hyoscine and gelsemium sometimes do good. Careful examination of the teeth, eyes, nose, stomach, and uterus for reflex irritation is imperative.

Galvanism, if carefully and persistently applied, almost always helps. It should be given daily. Various methods are described. The best way is to place the negative pole on the sternum or back of the neck, and the positive pole over each motor point of the nerve for one to two minutes, then over the occiput and over the facial cortical area for the same time. Currents of two to five milliamperes should be used. Neurectomy of the supra-orbital, continuous pressure on the motor points, stretching the nerve itself, are all measures which may be considered valueless.

Freezing the skin over the nerve with chloride of methyl has been recommended by Mitchell.

Blistering and cauterization are needless inflictions. The anæsthetization of the conjunctiva with cocaine is often helpful, both in diagnosis and treatment.

Blepharospasm is the name given to a tonic spasm of the orbicularis palpebrarum. It is generally caused by diseases of the eye, and its nature and treatment are matters belonging to ophthalmology. It is a rare symptom of hysteria.

Nictitating or winking spasm is a clonic spasm of the obicularis, and usually forms part of habit-chorea or is a symptom of hysteria.

Facial Palsies.

The paralyses of the facial nerve may be due to lesions that are central, nuclear, meningeal, or peripheral.

Facial palsy of *central origin* is almost invariably an accompaniment of hemiplegia and is due to hemorrhage, softening, inflammation, or tumor of the brain. The lower two branches of the facial are chiefly involved.

Facial palsy of *nuclear origin* is very rare and is an accompaniment of glosso-labial palsy, of diphtheritic palsy, or of gross lesions of the pons.

Facial palsy of *meningeal origin* is due to tumors, meningitis or fracture of the base of the brain and is accompanied by lesion of other cranial nerves. Syphilis is the most important factor here. All these forms are simply part of other diseases.

Peripheral facial palsy (Bell's palsy) is the common type of facial paralysis.

Etiology.—The typical cases of this disease are due to expo-

sure and rheumatic influences. Males are oftener affected, and the common age is between twenty and forty.* It is more frequent in the winter and in temperate climates. It is not hereditary. A neuropathic tendency and the rheumatic diatheses predispose to it. Syphilis rarely causes an isolated facial palsy; in fact, it is apt to leave this nerve alone (Hutchinson).

Facial palsy may occur in multiple neuritis and in locomotor ataxia. Non-typical and accidental cases of peripheral facial palsy are due to injuries, fracture of the petrous bone, or ear disease. Forceps pressure in difficult labor causes some cases, and a very few have been congenital.

Symptoms.—The disease comes on rather suddenly, and reaches its height within a few hours or, at most, two or three days. Preceding and accompanying the onset there may be some pain about the ears and a little swelling is sometimes seen.

The patient feels a subjective discomfort on the paralyzed side of the face. He finds that he cannot completely shut the eye; if he tries to chew on the affected side, food gets between the teeth and cheek. He cannot pucker the lips, and his speech is a little muffled. The appearance of the face is most characteristic.

On the affected side the wrinkles are smoothed out, the angle of the mouth is lower, the mouth is drawn at first to the sound side, and owing to this distortion the tongue appears not to be protruded straight. In laughing or other emotional movements of the face, the trouble is most clearly brought out. But the most characterized appearance is produced by telling the patient to shut the eyes tightly and draw out the angles of the mouth so as to show the teeth. The eye on the palsied side is not closed, and the eyeball turns up, showing the white of the eye. This test of the palsy is better than any examination of the wrinkles and folds of the face, for in children and in the young and plump these differences in the two sides of the face are not very marked, especially in the slighter cases.

The nostril on the affected side does not expand on forced inspiration; the eye is apt to be watery and the conjunctiva somewhat injected.

If the disease extend well up into the Fallopian canal so as to involve the nerve to the stapedius, that muscle is paralyzed, the tensor tympani acts unopposed, the drum is tightened, and unusual sensitiveness to sounds results. This is rare, however; most of the ear symptoms in facial palsy being due to a concomitant disorder of the tympanum or the acoustic nerve.

If the disease involve the nerve between the geniculate gan-

*Among 14 cases there were 9 males, 5 females. The ages were: Fifteen to twenty, 2; twenty-one to thirty, 7; thirty-one to forty, 3; forty-one to fifty, 2. Left side, 9; right, 5.

glion and the point where the chorda tympani is given off (see Fig. 68), some loss of taste follows, and this is a frequent symptom, but not one of long duration. If the disease is located more centrally than the geniculate ganglion or more peripherally than the chorda nerve, taste is not involved. Usually, when there is no taste involvment, it is because the lesion is peripheral. By an examination of the taste sense and of the hearing, the location of the trouble can be accurately made out.

After a few weeks some wasting of the face may be noted, but this is never very great.

The electrical reactions are characteristic and important, since typical or partial degeneration reactions can usually be observed. For a few days there is an increase in irritability of the muscles to both faradic and galvanic currents. This is followed by a gradual loss or diminution in faradic irritability, while the galvanic irritability remains sometimes increased for a while and then falls. At the same time a reversal of poles and a sluggish contraction, particularly the latter, may be noted. At the end of five or six weeks faradic irritability ought to begin to return. Great variations are found in the electrical reactions dependent upon the severity of the case.

If the disease lasts for two months or more and the palsy is not a complete one, secondary contractures begin to appear. The mouth now becomes drawn to the affected side, and the nasolabial fold becomes deeper than that on the sound side. In smiling or speaking or other facial movements there is an excess of movement on the paralyzed side, the teeth and upper gums in particular showing abnormally.

Pathology.—The disease in its typical form is due to a diffuse neuritis of rheumatic character. This attacks the periphery of the nerve in the face and extends rapidly up into the Fallopian canal as far as the geniculate ganglion. The inflammation sometimes attacks most the peripheral filaments; at other times it is more central. The old idea that it was always a perineuritis of the nerve in the Fallopian canal is incorrect (Minkowski).

Diagnosis.—The recognition of the palsy is made easy by causing the patient to contort the face. In children it requires more care to detect the side affected.

It is important to determine whether the palsy is cerebral, nuclear, basilar, or peripheral. If the cause is cerebral, the upper branch of the nerve is little affected and the *patient can close the eye.* The nerve and muscles show no degenerative reactions.

Nuclear palsy is very rare and is accompanied with other symptoms, especially those of involvement of other cranial nerves. A history of diphtheria, lead palsy, or bulbar paralysis is obtained.

In palsies due to lesions at the base of the brain such as gummy meningitis, the auditory and other cranial nerves are involved and there are signs of brain syphilis. By testing the sense of taste and hearing the location of the peripheral trouble can be made out (Fig. 68). Thus if there be loss of taste on the anterior two-thirds of the tongue, the lesion must be between the geniculate ganglion and the point at which the chorda tympani is given off. If the taste be not involved, the lesion must be central or peripheral of the part of the nerve which includes the chorda. Practically in most cases it is peripheral to it. If central, the disease is usually of syphilitic or tubercular origin; the palsy is severe and the loss of ability to close the eye very great.

Prognosis.—The prognosis of peripheral palsy (Bell's) is good, although an absolutely complete recovery often does not take place.

In syphilitic cases the prognosis is not so good, though patients often recover. In central palsies the prognosis is the worst, because the lesion usually does not disappear. However, the central cases are from the beginning of a mild type, and give annoyance mainly from the secondary contractures.

Bell's palsy usually lasts three to five months. Occasionally there are mild cases that get well within a month. The prognosis as to duration is much helped by a close study of the electrical reactions. In proportion as the degeneration reaction is complete and persistent the outlook is bad.

Treatment.—In the acute peripheral cases the patient should be treated promptly and thoroughly. He should be given a diuretic and purgative and a blister should be placed over the exit of the nerve. This should be followed or accompanied by hot fomentations. Salicylate of soda in full doses of twenty grains should be given during the first week. After the paralysis is established, iodide of potassium is indicated in moderate doses. Electricity is to be employed with care at first. After a week it may be given daily for five minutes, using the galvanic currents just strong enough to contract the muscles. After three or four weeks, if the faradic current causes contraction, it may be used, otherwise the galvanic current is to be continued. At the end of a month an application every other day is sufficient. At the end of three weeks, if the paralysis is severe, the corner of the mouth should be drawn up by means of a bent hook, which is carried back and fastened behind the ear. The patient should wear this most of the day-time, but not at night. The object is to take off the strain caused by the pulling of the muscles on the sound side.

At the end of a couple of months, if secondary contractures

set in massage may be tried, and the patient should practise facial gymnastics daily.

Acupuncture combined with weak faradic currents and local injections of strychnia may be tried in obstinate cases.

If the eye cannot be closed it should be protected by a shade.

DISEASES OF THE EIGHTH NERVE.

The diseases of the eighth or acoustic and space-sense nerve which are of especial interest to the neurologist are auditory paralysis or nervous deafness, hyperacusis, tinnitus aurium, and auditory vertigo or Menière's disease. These diseases correspond with loss, excess, and perversion or irritation of function.

Nervous Deafness (Acoustic Paralysis).

Nervous deafness may be due to lesions (1) of the cortical centres of hearing, (2) of the acoustic nuclei, and (3) of the acoustic nerve itself or its end-organ. Practically it is almost always the nerve and the internal ear which are affected.

Etiology.—1. Cortical nervous deafness has been known to occur in a few instances from lesions of both temporal lobes (Mills). A lesion in the left temporal lobe causes a form of sensory aphasia called word deafness. A lesion in the right temporal lobe may cause some deafness in the left ear. The deafness of hysteria is of cortical orgin.

2. Deafness from lesion of the acoustic nucleus or nerve root.

Cerebro-spinal meningitis in the young and syphilitic meningitis in the adult are the more frequent causes of this form. Tumors and hemorrhages may also be causes. When the auditory nucleus and nerve root are affected by these diseases, its peripheral terminations in the labyrinth are also often involved, so that sharp distinctions cannot always be drawn between this form and that due to labyrinthine disease.

3. *Deafness from Labyrinthine Disease.*—The causes are drugs, such as quinine and the salicylates; inflammations, including syphilitic exudates, injuries, hemorrhages, tumors, primary atrophy which may occur in locomotor ataxia; mechanical causes, such as the constant noises and jarring to which locomotive engineers and boiler-makers are subject.

Symptoms of Nervous Deafness.—The dominant symptom is loss of hearing, but this may be accompanied with vertigo, tinnitus, and even forced movements.

In hysterical deafness the loss of hearing is rarely complete, is unilateral, and especially involves high and low notes. Deafness from involvement of the nucleus and nerve root is usually accom-

panied with other symptoms of a basilar meningitis or lesion of the pons and medulla. Labyrinthine deafness is often associated with vertigo, tinnitus, and forced movements, when it may become a symptom-complex known as "Menière's disease."

Sudden total deafness is characteristic of syphilitic disease of the internal ear. In genuine nervous deafness, unless the deafness is absolute, bone conduction is lost, while aërial conduction is preserved. A tuning-fork vibrating on the skull or mastoid is not heard by the affected ear, though it is heard when held in the air close by this ear. Changes of reaction to the electrical current occur, but the tests are difficult and the results unsatisfactory.

The *treatment* depends upon the seat and nature of the lesion. In labyrinthine deafness it is generally limited to the use of iodide of potassium, mercury, pilocarpine, leeches, and the galvanic current. Local applications and surgical interference may be required. The treatment of nervous deafness in which vertigo, forced movements, and tinnitus are the dominant symptoms will be discussed under these heads.

Tinnitus Aurium, Tinnitus Cerebri (Noises in the Ear and Head).

Subjective sounds resembling hissing, buzzing, humming, beating, musical notes, etc., are classed together under the general head of tinnitus aurium. It is a very common symptom.

Etiology.—The disease attacks adults in middle and later life. Men and women are alike affected. Neuropathic constitutions and an unstable circulation favor it. The arterio-sclerosis of old age, cerebral anæmia and congestion, sunstroke, tobacco, and alcoholism lead to it. It occurs often in melancholia and in neurasthenia. Some local disease or congestion of the middle or internal ear is usually present. Tinnitus occurs in Bright's disease, gout, and dyspepsia.

Disease of the auditory nuclei and auditory tracts rarely, if ever, causes tinnitus; but chronic pachy-meningitis, such as follows blows on the head, sunstroke, alcoholism, etc., may be attended with most annoying tinnitus, which is often not so much in the ears as in the head—a tinnitus cerebri. In old people with thickened arteries and imperfect brain nutrition a similar condition may occur.

Tinnitus accompanies insanity sometimes, and may be the source of aural hallucinations.

A kind of tinnitus may accompany migraine and take the form of an aura in epilepsy.

Despite this long list of causes, the chief factors may be

summed up as neurasthenic states, local ear disease, humoral poisons and irritants, reflex irritants, arterio-sclerosis.

The *symptoms* are indicated by the name of the malady. They may come on suddenly, but usually develop slowly. Some deafness and occasional vertigo are often present. The noise is located in one ear as a rule. Sometimes it is said to be simply "in the head." The sounds are generally present all the time, giving the patient little rest and making life a burden. They vary greatly in character and intensity. These variations are indicated in the study of the diagnosis.

The Diagnosis.—The recognition of the symptom is easy. The principal thing is to discover its seat and cause.

The ear should of course be examined for external or middle ear disease.

If the tinnitus is pulsating and synchronous with the heart-beats and stopped by carotid compression, it may be inferred that it is due to vasomotor paralysis, or inflammatory congestion, or aneurism.

If the sound is not in the ear, but in the head, and not associated with deafness or ear disease, the trouble is probably central, and most likely is of meningeal or arterio-sclerotic origin.

Noises which are complex or take the form of musical sounds or words are probably central.

Constant rushing, knocking, pulsating noises are due to congestion, hemorrhage, or inflammatory effusion in the labyrinth.

Moist sounds of a gurgling, bubbling, boiling, singing, whistling, shell-like roaring character indicate disease of the middle ear, with fluid exudation or catarrh of the Eustachian tube, or irritation of the external auditory canal, mastoid cells, or post-nasal spaces.

Dry roaring and ringing noises are due to non-suppurative catarrh of middle ear, disease of the muscles or nervous supply of the tympanum, meningitis, tumors, and syphilis.

The condition of the digestion should be inquired into and the existence of renal, arterial, or of central nervous disease investigated.

Treatment.—Hydrobromic acid and the other bromides are the surest remedies for this trouble, given in ordinary doses. They may be combined with digitalis. Iodide of potassium and iodide of ethyl often are useful. Nitroglycerin is sometimes of value in patients with hard arteries. A combination of digitalis, bromide, and nitroglycerin has given me the best results. Occasionally tonics are indicated. I have seen but little good from electricity or counter-irritation. Of course middle-ear disease must be treated if it is present.

Vertigo (Dizziness, Giddiness).

Vertigo is a disturbance of consciousness characterized by apparent movements of external objects or of the person himself. If external objects whirl around, the vertigo is called *objective;* if the person himself seems to move, it is called *subjective.* Vertigo is almost always a symptom. In rare cases it appears to be *idiopathic.*

Vertigo is connected more or less with our space sensations, and hence it will be described here under disorders of the eighth cranial nerve and its central representations.

Etiology.—The causes of vertigo may be classed somewhat like those of headache, as follows:

1, Hæmic: anæmia, hyperæmia, toxæmia, from tobacco and alcohol; 2, arterio-sclerosis; 3, acoustic-nerve irritation; 4, neuroses: epilepsy, neurasthenia; 5, reflex: ocular, gastric; 6, organic brain disease; 7, mechanical causes like electricity, swinging, etc.

Based mainly on the etiology, we have as a practical classification of ordinary cases of vertigo: toxic, auditory, gastric and bilious, ocular, neurasthenic, and epileptic forms. The various causes of vertigo act partly by irritating the space-sense nerve and thus disturbing our sense of relation to external objects, partly by irritating the cortical centres of the brain.

Symptoms.—Vertigo comes on suddenly, and lasts, as a rule, for but a moment. The floor rises up and sinks down, or objects whirl around (objective vertigo), or the patient seems whirling around or falling. The ideas are confused, there is a sense of alarm and a feeling of faintness. The patient totters, sometimes falls; there may be nausea or vomiting. In some forms there is momentary loss of consciousnses, or syncope. Vertigo usually comes on in short attacks, but in toxæmic states as in alcoholism or nicotinism it is almost constant while the poison is in the system.

Vertigo may become chronic or nearly so; and if severe it forms what is called the *status vertiginosus* (Mitchell). Vertigo is increased by rising or sudden movements and lessened by lying down.

Vertigo due to organic lesions of the cerebellum and its peduncles, or of the labyrinth, is associated with forced movements of the body.

Symptoms of Special Forms.—Auditory Vertigo (Menière's Disease).—A large proportion of vertigoes are due to disease or irritation of the eighth nerve and its centres. The common cause is local disease of the labyrinth. When this produces severe at-

tacks of vertigo with nausea and perhaps syncope, it is called "Menière's disease." The name is often applied to any form of auditory vertigo. Menière's type is always due to organic disease of the labyrinth. It is accompanied with progressive deafness, and sometimes with tinnitus and forced movements, or even an utter inability to walk steadily. When the deafness is complete the vertigo ceases, because the nerve end-organ is destroyed. Mild forms of auditory vertigo present nothing unusual except those due to involvement of the nerve of hearing.

Many forms of vertigo, such as the gastric and toxic, occur through a reflex disturbance of the eighth nerve. The auditory nuclei are connected with those of the vagi. The labyrinth is supplied with blood by the vertebral artery, whose calibre is controlled by sympathetic fibres which are in close connection with fibres to the stomach. Hence reflex effects may occur through contiguity of the central nuclei and by reflex spasm of the vessels of the internal ear. "Stomachal vertigo" is the name given to a very severe form of reflex vertigo. It occurs generally in persons whose stomachs are overloaded and digestion paralyzed by its load. It is accompanied by loss of consciousness.

Bilious and Lithæmic Vertigo.—In conditions of dyspepsia, constipation, and hepatic torpor, the disordered stomach and bowel suddenly discharge into the blood irritant substances which pass to the brain and by direct action on the nervous centres cause vertigo. This is probably the explanation of the vertigo of biliousness and constipation. It is a paroxysmal vertigo, noted most in the morning, not very severe, and often accompanied with nausea.

Neurotic Vertigo.—The symptoms of epileptic vertigo will be described under that head.

Neurasthenic vertigo is a not uncommon symptom. The attacks are short, generally subjective, not severe or accompanied with nausea or syncope, but they often cause much alarm. Underlying them are exhausted and irritable nerve centres, with ocular, gastric, and humoral irritations.

A neurotic vertigo occurs sometimes in the form of attacks almost exactly resembling seasickness. There is intense vertigo, nausea, and faintness lasting for hours, coming on suddenly without known cause except overwork or excitement. The attacks occur in neurotic subjects and are analogous to other nervous crises. It is a periodical neurosis of the space-sense nerve.

In some nervous subjects there occurs a sudden giving way of the legs. There is no conscious vertigo, yet such probably exists. The symptom is noted in exophthalmic goitre. It is a "stumbling vertigo."

Ocular vertigo is a rare symptom, but is, when present, chronic

and annoying. It is caused by refractive errors and unequal action of the ocular muscles.

The *mechanical vertigoes* are such as are produced by swinging, whirling, the movements of the ship, steam-car, and elevators. Railway mail-clerks, elevator boys, often suffer from chronic disturbances of a vertiginous character. Ocular and auditory nerve sensations enter into the causation.

Arterio-sclerotic Vertigo, Senile Vertigo.—This occurs in persons who have arterio-sclerotic changes in the brain-vessels, either from disease or senility. The symptom is caused by impaired brain nutrition with consequent anæmia. Senile vertigo may also be due to a weak and fatty heart.

Pathology.—The consciousness of the proper equilibrium of the body and of its relations to the external world depends upon the continuous inflow of nervous impulses from the eye and its muscles, from the nerves of the muscles, joints, and viscera, and from the ear. Anything which suddenly disorders this even inflow may cause a disturbance of consciousness and sensations of vertigo.

The aural impulses come from the semi-circular canals and ampullæ; they are the most important. These impulses are not felt in consciousness normally, but go to certain lower centres chiefly in the vermis of the cerebellum. From this point they influence the acts concerned in holding the body in equilibrium.

When impulses from the eye and its adjusting mechanism do not flow in normally, there may be disturbance of consciousness and a feeling of vertigo. Probably visceral impulses can produce a similar disturbance. Everything which suddenly interferes with the nutrition of the cortex of the brain, such as anæmia and poisons, may lead to giddiness by lowering the level of consciousness and confusing the sensory inflow.

Diagnosis.—In investigating vertigo the physician should find (1) whether it is subjective or objective, (2) paroxysmal or chronic, (3) accompanied with ear symptoms, nausea, tinnitus, and loss of consciousness. He should then direct himself to finding the special cause and seat, remembering that the auditory, gastric, toxic, and neurasthenic are the common forms. In elderly persons the arteries should be carefully examined. In young persons the possibility of epilepsy must be remembered.

The *prognosis* depends upon the cause. Epileptic vertigo and vertigo from organic disease are most serious. Labyrinthine vertigo usually ceases when complete deafness occurs. The other forms of vertigo are usually susceptible of relief.

Treatment.—The attack is treated by rest in the horizontal position and the administration of a volatile stimulant. The disorder must then be treated in accordance with the cause.

In Menière's vertigo the use of quinine by Charcot's method is said to be useful. Quinine is given in doses which are gradually increased until cinchonism results. Then the drug is stopped. Mitchell advises the addition of hydrobromic acid; Gowers advises the use of salicylate of soda in five-grain doses instead of quinine. Hirt recommends ten drops of a two-per-cent solution of pilocarpine injected hypodermically every other day.

Neurasthenic vertigo is cured by rest and attention to diet, laxatives and mineral acids being used. Hydrobromic acid with pepsin and glycerin are often very helpful here. Gastric vertigo is to be treated with saline laxatives and simple bitters before meals.

In the vertigo of "biliousness" and lithæmia there is often a neurasthenic element, and a similar attention to diet and to the digestive organs is indicated. In arterio-sclerotic and senile vertigo small doses of nitroglycerin and iodide of potassium, with or without digitalis, should be given. Rest and warmth of the extremities are indicated. In all forms of vertigo bromide of potassium is helpful and will relieve the symptoms for a time. It is the best symptomatic remedy. Counter-irritation to the neck or mastoid region by the cautery does good occasionally.

There are two peculiar forms of disease to which the name vertigo has been attached which may be described here.

Laryngeal Syncope (Laryngeal Vertigo, L. Epilepsy).—This is a rare form of disorder characterized by attacks of paræsthesia of the throat, with coughing, followed by sudden syncope, and sometimes by slight convulsive movements.

The disease occurs chiefly in males at about the age of fifty, though the range of age is thirty-five to seventy. Neurotic constitution is often present. There may be a history of injury and the use of stimulants. Laryngitis, bronchitis, apical phthisis, and asthma may be present.

At the onset of the attacks a burning or tickling sensation is felt in the larynx or trachea; there is a spasmodic cough, perhaps some asthmatic or dyspnœic symptoms, when the patient suddenly falls, unconscious for a short time. The attacks may occur daily or only once in a few weeks. Most cases are curable, yet the disease is not without danger.

It is probably a reflex neurosis, not a true epilepsy.

The treatment should be directed to the relieving any local condition or pulmonary trouble. Bromide of potassium should also be given.

Paralyzing Vertigo (Gerlier's Disease).—This is a disease occurring only on the farms in southern France and Switzerland.

The symptoms consist of sudden attacks of ptosis, vertigo, paresis of arms and legs, and cervico-occipital pain. The disease is most prevalent in the summer-time. It attacks chiefly males. Single attacks last not over ten minutes, but may occur frequently.

The cause is supposed to be a special microbe developed in the stables during the heat of summer.

We are not aware of its occurrence in America, though Seguin calls attention to the close similarity of the symptoms to those of poisoning by conium maculatum.

HYPERACUSIS (AUDITORY HYPERÆSTHESIA).—When there is undue keenness of the sense of hearing, the condition is called hyperacusis. It occurs in hysteria and hypnotic states. Some persons have naturally an extraordinary keenness of hearing. In facial paralysis there is sometimes hyperacusis due to paralysis of the stapedius.

When ordinary sounds cause painful feelings, the condition is called dysacusis. This occurs in the neurasthenic and hysterical, in persons of enfeebled vitality, in the brain congestion of fevers, and in meningitis; also in local ear troubles of an inflammatory character.

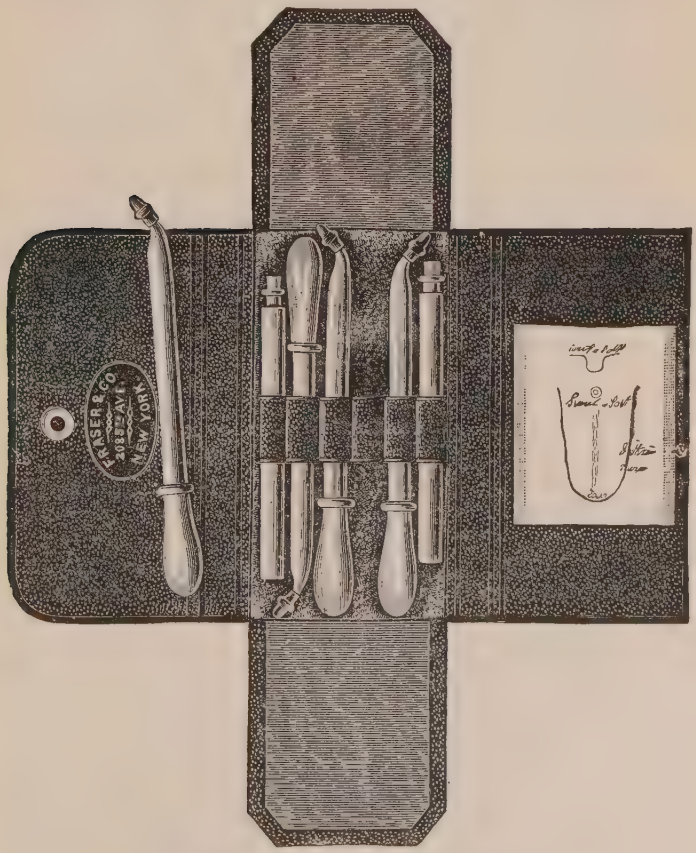


FIG. 67.—CASE FOR TESTING SENSE OF TASTE AND SMELL.

Diseases of the Glosso-Pharyngeal Nerve.

The motor fibres are affected with spasm causing dysphagia and a feeling called globus; they are also affected by paralysis in the disease called glosso-labio-laryngeal palsy.

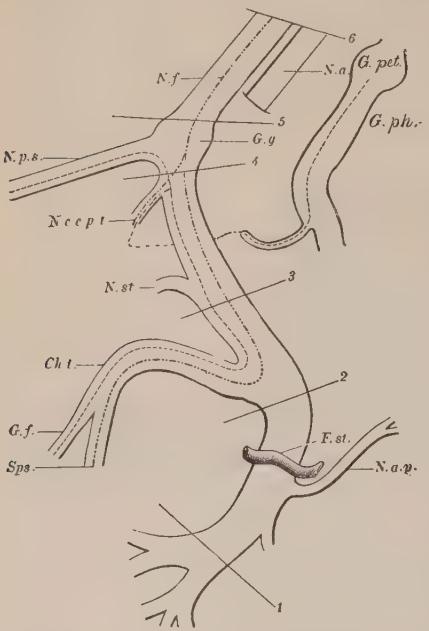


Fig. 68.—Modified from Erb, showing the Relations of the Seventh and Glosso-Pharyngeal Nerves and the Course of the Taste Fibres. The lines 1, 2, 3, 4, and 5 mark off different segments of the nerve; *N.f.*, facial nerve; *N.a.*, acoustic nerve; *G.ph.*, glosso-pharyngeal nerve; *G.g.*, geniculate ganglion; *N.p.s.*, great superficial pelvosal from fifth nerve; *N.c.c.p.t.*, tympanic plexus; the dotted line *Sps.* indicates salivary fibres; the other dotted line indicates taste fibres; *Ch.t.*, chorda tympani; *N.st.*, stapedius nerve; *F.st.*, stylo-mastoid foramen.

The sensory fibres are affected also in globus. The special sense of taste is affected, causing ageusia or loss of taste.

Of these diseases no one belongs to the glosso-pharyngeal nerve alone. It will be convenient, however, to describe disorders of taste here.

Ageusia (loss of the sense of taste) is an affection in which the power to discriminate the tastes of bitter, sweet, salt, acid, and alkaline substances is lost.

Etiology.—It occurs oftenest in an incomplete form in facial palsy and in hysteria. Injuries of the trigeminus and glosso-pharyngeal nerves, catarrhal diseases of the mucous membrane of the mouth and nose, are frequent causes. It is not caused by organic brain disease so far as known.

Symptoms. — Some ageusia is present in the imbecile, and the sense of taste is less keen in the lowly organized and criminal classes.

The symptoms are subjective and may not be noticed at first by the patient. In hemiageusia from facial palsy and hysteria it has to be looked for, as the patient does not complain. The tests are made with solutions of salt, sugar, vinegar, and quinine. A

single solution of sugar usually answers. But the different parts of the tongue differ in sensibility to different substances (Fig. 67).

Care must be taken to exclude the nose as a factor in taste.

Pathology.—Ageusia occurs as the result of disease of the roots of the trigeminus, especially of the second, also from disease of the facial when the chorda tympani is implicated and from disease of the glosso-pharyngeal root.

Disease of the trigeminus and facial usually causes ageusia on the anterior two-thirds of the tongue, with loss of taste, especially for sour and bitter substances. Sometimes, however, disease of the trigeminus or disease of the tympanum involving the tympanic plexus and chorda tympani causes ageusia of the whole tongue on the affected side.

Ageusia from disease of the glosso-pharyngeal alone is very rare, and then causes loss of taste on the posterior third of the tongue, soft palate and pillars of the fauces, with loss of taste to sweets and acids. A few cases have been reported in which paralysis of the glosso-pharyngeal caused complete ageusia on the affected side. It must be inferred, therefore, that taste fibres run sometimes wholly in the fifth, more rarely wholly in the ninth nerves, and usually in both.

The *treatment* depends on the cause. Locally, cleansing and stimulating mouth-washes and electricity may be used.

Parageusia or perversions and imperfections in the taste sense are very frequent. They are generally due to irritation of the taste nerves from catarrhal inflammation of the stomach or mouth. They also occur in hysteria.

Diseases of the Pneumo-gastric Nerve and of the Bulbar Portion of the Spinal Accessory.

These nerves are essentially visceral in character. Their diseases call for a study of laryngeal, pulmonary, cardiac, and abdominal neuroses, which would bring us into the domain of laryngology and general medicine. Hence, despite their great importance, I have thought it best not to try and present them here. Some of the symptoms are described in connection with locomotor ataxia, progressive muscular atrophy, exophthalmic goitre, and angina pectoris.

Diseases of the Hypoglossal Nerve.

The diseases of this nerve consist of lingual spasms, lingual palsy, and lingual hemiatrophy.

Lingual spasms take part in the disorders of articulation, helping to cause stuttering and speech cramps. Such troubles are

often developmental in origin and belong to the habit-choreas or convulsive tics.

Stuttering is a spasmodic disorder in which the tongue muscles are involved, preventing the proper enunciation of words and sentences.

Stammering is an imperfect articulation due sometimes to disease or defect in the hypoglossal nerve and its muscles. It is not a spasm.

Aphthongia is the name given to a form of spasm occurring in speakers and similar in nature to writer's cramp.

Clonic lingual spasm occurs in chorea, hysteria, and during the attacks of epilepsy. Unique cases of this spasm also occur from reflex irritation or central nervous disease.

Tonic lingual spasm occurs in hysteria, and sometimes as an independent affection due to unknown causes, generally those of a debilitating and nervously depressing character. Reflex irritation may be a cause.

Lingual paralysis (glossoplegia) is usually one of the symptoms of glosso-labio-laryngeal palsy. It may be caused by a bilateral lesion in the cerebral hemispheres. The condition is then known as pseudo-bulbar paralysis. Diseases of the medulla and of the nerve itself may cause the paralysis.

The paralysis may be either unilateral or bilateral. The symptoms are an impairment of speech and of swallowing. Fuller details will be given under the head of Bulbar Palsy.

Progressive Lingual Hemiatrophy.—A progressive hemiatrophy of the tongue sometimes occurs. It is analogous in all respects to facial hemiatrophy, with which it is sometimes associated. It is probably due to a low grade of degenerative neuritis of the nerve. It is very rare.

DISEASES OF THE SPINAL PART OF THE ACCESSORIUS.

This is a purely motor nerve, and its disorders are therefore spasm and paralysis.

Torticollis (Wry-Neck, Caput Obstipum).

Torticollis is a disease characterized by clonic or tonic spasm of the muscles supplied by the spinal accessory and often of other muscles of the neck. There are several forms of wry-neck, which must be distinguished from each other. They are: 1, congenital wry-neck; 2, symptomatic wry-neck; 3, spurious wry-neck, from spinal disease; 4, true spasmodic wry-neck.

1. *Congenital wry-neck* is due to some intra-uterine atrophy or obstetrical injury of the sterno-cleido-mastoid. It occurs oftenest after breech or foot presentations. The right side is

usually affected. There is no spasm at all, but the neck is fixed to one side by the shortness of the muscle, and also rotated to the opposite side. The deformity becomes more noticeable as the child grows older, because the parts atrophy. The atrophy affects not only the shortened muscles, but the face on the affected side.

2. *Symptomatic wry-neck* is usually due to a rheumatic myositis, and occurs chiefly in children. It may be due also to tumors, adenitis, abscesses, and local syphilitic disease. In these cases there is always pain and tenderness associated with the deformity.

3. *Spurious wry-neck* is an apparent or real spasm of the neck muscles due to caries of the spine.

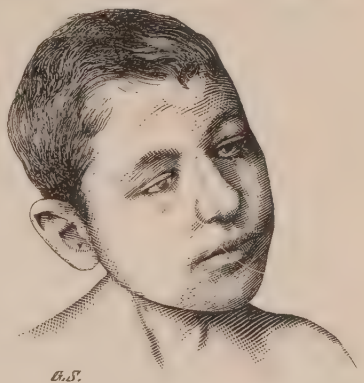


Fig. 69.—Wry-Neck of Right Side.

Treatment.—Congenital wry-neck, if taken early, can be cured by tenotomy of the sterno-mastoid and subsequent fixation of the neck for a time. When osseous changes have occurred perfect relief is impossible. Symptomatic rheumatic wry-neck is a trivial and temporary affection, which needs only to be palliated by hot applications and saline purges until cure takes place.

Spurious wry-neck requires suitable orthopedic measures, such as the plaster jacket and jury-mast.

4. *Spasmodic Wry-Neck.*—This is a purely nervous disease characterized by spasm of the muscles supplied by the spinal accessory and sometimes by the upper cervical nerves.

Etiology.—Women are much oftener affected than men. It occurs in early adult and middle life, never in children or old people. A neuropathic constitution and heredity often exist.

The exciting causes are occupations which put the lateral muscles of the neck on a strain, depressing emotions, physical shocks and blows, rheumatic influences, and perhaps malaria. Sometimes no cause can be detected. Reflex irritations, perhaps, exist in some cases, but it is difficult to find them.

Symptoms.—The disease begins with slightly painful sensations in the neck, which are soon accompanied with spasm. The spasm is at first clonic and intermittent. The sterno-mastoid is oftenest involved of single muscles; but the rule is that the upper fibres of the trapezius are also affected. The patient's

head is inclined toward the affected side by the trapezius, the chin is raised and the head rotated to the opposite side by the sterno-mastoid and trapezius, and this is the typical position in the disease. If both trapezii are affected the head is pulled back, but this is a rare form. It is called retrocollic spasm. The complexus and obliquus superior are the only other neck muscles which can rotate the head to the opposite side. They are supplied by the upper cervical nerves and are sometimes involved in wry-neck. In torticollis the muscles affected with spasm have a similar physiological function. Hence while the sterno-mastoid, trapezius, complexus, and superior oblique on one side are attacked by the spasms, muscles on the other side may be at the same time implicated. The opposite muscle commonly affected is the splenius, which inclines the head laterally and rotates it to the same side. Probably the deep muscles, recti capitis postici, major and minor, and the inferior oblique, which draw the head back and rotate to the same side, are also at times affected. The list of muscles that may be involved and their nerve supply is as follows:

	Function.		
	Turning Head to Opposite Side.	Turning or Inclining Head to Same Side.	Nerve Supply.
Muscles usually involved.	Sterno-cleido-mastoid	....	Eleventh.
	Upper fibres of trapezius..	.....	Eleventh.
Muscles rarely involved.	Superior obliquus.....	Recti capitis postici, maj., min., infer. obliq., splenius.	Cervical.
	Complexus		Cervical.

Extreme rotation without much retraction of the head would indicate involvement of the sterno-cleido-mastoid and opposite splenius. Retraction of the head indicates involvement of both trapezii.

The disease may start in one muscle and gradually extend to others, even involving the facial, masticatory, and brachial nerves. As it progresses the spasm becomes more constant, and finally it may be tonic, never yielding except to artificial means or during sleep. The pain associated with the disease gradually decreases. The affected muscles hypertrophy, the muscles thrown into disuse atrophy. There is some deformity, in time, of the neck and shoulders, but facial asymmetry does not occur in this form unless it begins, as is very rarely the case, before maturity.

The disease may be complicated with or alternate with other neuroses. I have known epilepsy to be associated with it.

Pathology.—The disease is a neurosis involving the bulbar and lower cerebral centres. The neuro-mechanism controlling the movements of the neck is unstable and out of control. Conse-

quently it sends out intermittent and irregular discharges of nerve force. The seat of the typical disease is never in the nerve alone, probably not in the cortex.

It is a disturbance of nutrition and function; it has no pathological anatomy, though vascular disturbances of the nerves and centres may be present.

The *diagnosis* has to be made from the other forms of wry-neck mentioned. The age, history, and fixed character of the spasm serve to distinguish congenital wry-neck. The history, the pain and tenderness, and the temporary duration differentiate the rheumatic forms.

The increased rigidity on passive motion, the pain, deformity, and other signs of cervical caries are sufficient to diagnosticate vertebral disease.

Prognosis.—The disease is not fatal. It generally reaches a certain stage and then remains chronic. In rare cases it is cured; in many others it can be much ameliorated. Cases occurring in young people, in the hysterical, and in those without a decided neurotic history are the most favorable.

Treatment.—The drugs which are efficient are opium, atropia or hyoscyamine, conium, gelsemium, valerianate of zinc, asafœtida, chloral, bromides, arsenic, and cocaine. Of these, opium, atropia, gelsemium, and zinc are generally the most efficacious. Opium must be given with great caution. Atropia should be given hypodermically in increasing doses up to intoxication (gr. $\frac{1}{10}$) (Leszynsky). The galvanic and faradic currents are useful adjuvants in helping to relax the spasm and keep up the nutrition of the muscles; but alone they are not curative. Massage and stretching the neck in a Sayre apparatus, together with systematic exercise of the neck muscles, often help; neck-stretching will even sometimes cure. The only surgical measures to be advised are nerve-resection, and possibly the partial cutting of the sterno-mastoid muscle. A very few cures and many failures have followed surgical interference.

Splints and mechanical fixation apparatus do no good as a rule.

Resection of the posterior branches of the upper three or four cervical nerves as suggested by Keen has done great good in a few cases (Powers).

Spasmus Nutans (eclampsia nutans, nodding spasm, salaam spasm, oscillating spasm).—This is a disorder occurring chiefly in children and characterized by rhythmical nodding or oscillating movements of the head.

Etiology.—The disease occurs in young children who are anæmic and badly nourished. Dentition, digestive disorders, basilar meningitis, gross disease of the brain, are causes. Sometimes it is

only a kind of habit chorea, and occasionally this habit continues during life.

Symptoms.—The disease may come on suddenly; more rarely it develops slowly. The patient has paroxysms usually of a rather violent character, lasting for minutes or hours, or even continuing nearly all the time except during sleep. The head moves thirty to sixty times a minute usually, but the motion may be slower or faster. Movements of the eyes and facial muscles often complicate the affection. The paroxysm may end in an epileptic attack.

The *diagnosis* is easily made by the symptoms. The *prognosis* and *treatment* depend upon the etiology. Bromide of potassium, hyoscine, and syrup of iodide of iron I have found useful.

Paralysis of the Spinal Part of the Accessory.—*Etiology.*—The causes are injuries, caries of vertebra, progressive muscular atrophy, and all forms of spinal disease reaching high up in the cervical cord.

Symptoms.—When one nerve is paralyzed the head may still be held straight, but there is inability to rotate it perfectly. The prominence of the sterno-mastoid is absent—atrophy takes place. No spasm of the other muscle occurs, and there is no such thing as paralytic torticollis (Gowers). The involvement of the trapezius causes a depression in the contour of the neck, especially noticeable on deep inspiration. There is some trouble in raising the arm, the scapula is drawn away from the spine, and the lower angle is rotated inward. When both nerves are paralyzed there is great difficulty in rotating the head or raising the chin. Paralysis of both sterno-mastoids causes the chin to drop backward, while paralysis of both trapezii in their upper parts causes the head to drop forward. Atrophy of the muscles attends the paralysis of the nerve, and degenerative reactions may be noted. The cervical nerves appear sometimes to supply the sterno-mastoid and upper part of the trapezius so much that in disease of the accessories decided paralytic symptoms are absent. When both parts of the spinal accessory are involved, dropping of the palate, dysphonia, and rapid pulse are added symptoms.

The *diagnosis* depends upon a thorough examination of the motility of the parts.

The *treatment* is based on a knowledge of the cause of the disease.

CHAPTER IX.

DISEASES OF THE SPINAL NERVES.

Anatomy and Physiology.—The spinal nerves arise from the spinal cord by two roots, anterior and posterior. These roots unite outside the spinal canal to form mixed nerves. The mixed nerves divide and go to their various destinations. There are thirty-three pair of spinal nerves, viz.:

Cervical,	8	
Dorsal,	12	
Lumbar,	5	
Sacral,	5	
Coccygeal,	3	(all rudimentary)
	33	

The last two coccygeal nerves are microscopic in size, and the first pair is very small, so that practically there are but thirty sets of spinal nerves.

The posterior roots are connected with ganglia lying in the intervertebral canal, and called intervertebral ganglia, or ganglia of the posterior roots. The mixed nerve is connected, by fibres that come chiefly from the anterior root, with the sympathetic or prævertebral ganglia.

For the purpose of conveniently studying the diseases of the spinal nerves, we divide them into six different groups, each having a somewhat definite work to do. These groups are shown in the accompanying table, also on p. 82.

	Strands of Spinal Nerves.	Distribution.	Associated Ganglia of Sympathetic.
Group I....	Upper four cervical.	Occipital region, neck.	First cervical.
" II....	Lower four cervical and first dorsal.	Upper extremities.	Second and third cervical, first dorsal.
" III....	Upper six dorsal.	Thoracic wall.	First to sixth dorsal.
" IV....	Lower six dorsal except last.	Abdominal wall, upper lumbar, upper lateral thigh surface.	Fifth to twelfth dorsal.
" V....	Twelfth dorsal, four lumbar.	Lumbar region, upper gluteal, anterior and inner thigh and knee.	First to fourth lumbar.
" VI....	Fifth lumbar and five sacral.	Lower gluteal, posterior thigh, leg.	First to fifth sacral.

Group I. The Upper Cervical

includes the first four of the spinal nerves. These divide into anterior and posterior branches. The posterior branches supply the muscles and skin of the back of the neck and the occiput. The

principal nerves are the suboccipital and great occipital. The anterior branches form the cervical plexus. Its principal branches are the auricularis magnus, occipitalis minor, and phrenic. The

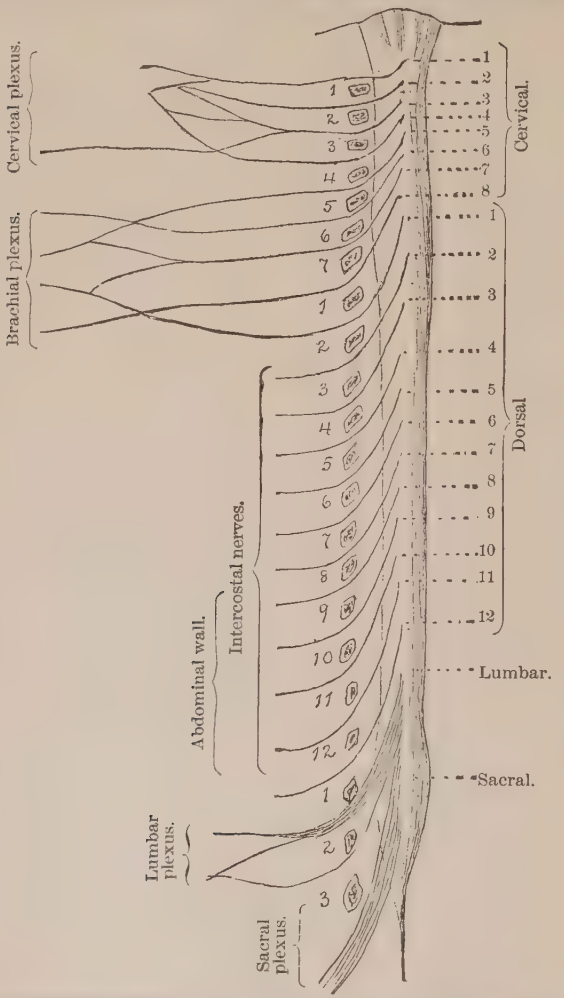


FIG. 70.—THE SPINAL NERVES, showing their groupings and plexuses (after Baker).

special distribution of the nerves is shown in the table and Fig. 71.

The upper cervical nerves supply motion to the muscles which rotate the head and draw it back and sideways.

One branch, the phrenic, supplies the diaphragm; other mus-

cles assist in fixing the thorax in forced inspiration. They innervate some of the hyoid and thyroid muscles, but have no influence on phonation or deglutition. This group of nerves is in close connection centrally with the trigeminal nerve, whose descending root reaches down into the cervical cord. The fibres to the scalp and face also anastomose with the trigeminus in their peripheral distribution to the scalp and chin.

The diseases of the upper cervical group are spasms, paralyses, and neuralgias.

Spasmodic Diseases.—Torticollis may be limited to the cervical nerves, as has been shown.

Tonic spasm causing a rigid neck is a frequent symptom of meningitis, and forms part of epileptic and other convulsions.

In oscillatory and rotatory spasms the cervical nerves are involved.

Hiccough is a clonic spasmodic disorder of the phrenic nerve. It is usually due to gastric disturbance, with flatulent distention of the stomach.

When chronic, it is caused by hysteria, neuritis, diaphragmatic pleurisy, or some pressure upon the nerve in its course. I have seen a case in which it was probably a pure spasmodic neurosis. Ordinarily, hiccough can be stopped by simple carminatives like spirits of chloroform or lavender. In obstinate cases in which no known cause can be found, pilocarpine, hyoscine, and bromides are useful. Counter-irritation along the neck helps some cases. A most effective measure is to lay the patient supine over a thick bolster so that the head hangs down and the thorax arches up.

Paralyses and Atrophy.—The cervical muscles are paralyzed in progressive muscular atrophy and in pachy-meningitis hypertrophica, and occasionally in vertebral and peripheral disease or injury. Some deformities and weakness in head movements result, but the most serious consequence is involvement of respiration through palsy of the phrenic.

Paralysis of the Phrenic Nerve.—*Etiology.*—Such paralysis may be due to disease or injury of the cervical cord, and also to peripheral disease, to which the nerve is somewhat liable owing to its long course through the anterior mediastinum.

Pleurisy, peritonitis, mediastinal tumors, rheumatic and toxic influences, and hysteria are among the special causes of phrenic paralysis. Spinal-cord disease is, however, the commonest etiological factor.

Symptoms.—In diaphragmatic paralysis, if bilateral, as is usually the case, there is either no movement of the abdomen or the epigastrium and hypochondrium are drawn in. On slight exertion there is dyspnœa and increase of respiration.

Diagnosis.—If no other muscles than the diaphragm are involved, the cause is probably in the trunk of the nerve. Inflammatory disease of the diaphragm may cause a paralysis which is recognized by its painful character and the febrile reaction.

Treatment.—This is to be guided by the cause. It need only be said here that there is a motor point in the neck where by

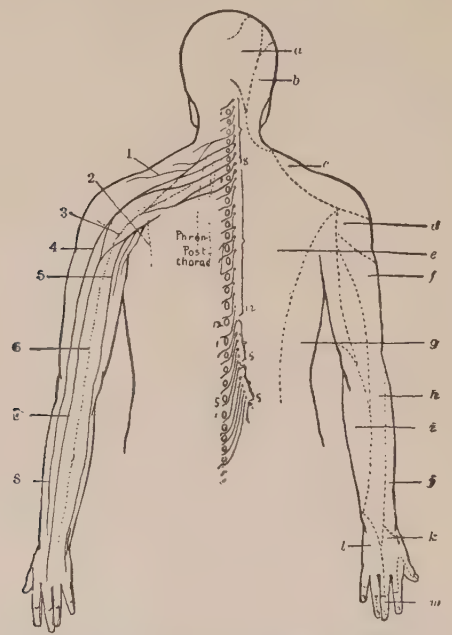


FIG. 71.—SHOWING: (1) The relation of the spinal nerve roots to the spinous processes; (2) the arrangement of the brachial plexus; (3) the level of origin and (4) the distribution of the chief motor and sensory nerves of the arm. Small letters refer to sensory distribution, figures to mixed nerves; capital letters represent the nerve roots from which the nerves are derived. *a*, Great occipital; *b*, small occipital; *c*, clavicular; *d*, circumflex; *e*, posterior dorsal; *f*, musculo-spiral; *g*, lateral dorsal; *h*, external cutaneous; *i*, internal cutaneous; *j*, musculo-cutaneous; *k*, radial; *l*, ulnar; *m*, median.

1. Suprascapular. (From 5 and 6 C.)

2. Subscapular. (From 5 and 6 C.)

3 and 6. Musculo-spiral. (From 4, 5, 6, 7, 8 C. roots.)
- Supinators.
- Ext. carp. rad. and ulnar.
- Ext. com. dig.
- Ext. pollicis.
- Triceps and anconeus.

4. Musculo-cutaneous. (From 4 and 5 C. roots.)
- Biceps.
- Brachialis ant.

5. Ulnar. (From 8 C. and 1 D.)
- Flex. carp. ulnar.
- One-half flex. prof. dig. interossei.
- One-half lumbricales.
- One-half flex. pollic.
- Add. pollicis.

7. Median. (From 6, 7, 8 C.)
- Pronators.
- Flex. carp. rad.
- Flex. subl. dig.
- One-half flex. prof. dig.
- Abd. oppon. and one-half flexor pollicis.
- One-half lumbricales.

careful electrization one can get a contraction of the diaphragm. In paralysis this fact should be utilized.

Cervico-Occipital Neuralgia—Neck Pains.—*Etiology.*—Pains in the back of the head and neck occur in migraine, in hysteria, spinal irritation, and neurasthenia, as a result of eye strain, as a true neuralgia, and as a symptom of brain tumor, meningitis, and rheumatic inflammation of the neck muscles and nerves. True cervico-occipital neuralgia is not rare, is much more common in women, occurs between the ages of twenty and thirty-five, and is often a reflex of pelvic disease.

Symptoms. — Migrainous pains have been described. In spinal irritation and hysteria the trouble is central, or perhaps shifting; it is especially characterized by a sharp boring pain just below the occiput. With it there may be evidences of cerebral congestion or anæmia, with vertigo and faintness, but not vomiting. The boring pain is almost pathognomonic of spinal irritation. In neurasthenia the pain is more of a tired, aching character. In typical neuralgia the pain is usually unilateral, paroxysmal, and sharp, sometimes reaching the intensity of a tic douloureux. There are tender points over the exit of the nerves. The disease lasts for five or six weeks. If of reflex origin, it may become chronic. The pains may alternate with or take the place of a trigeminal neuralgia. The nerves involved are the great and small occipital from the second pair and a branch from the third pair.

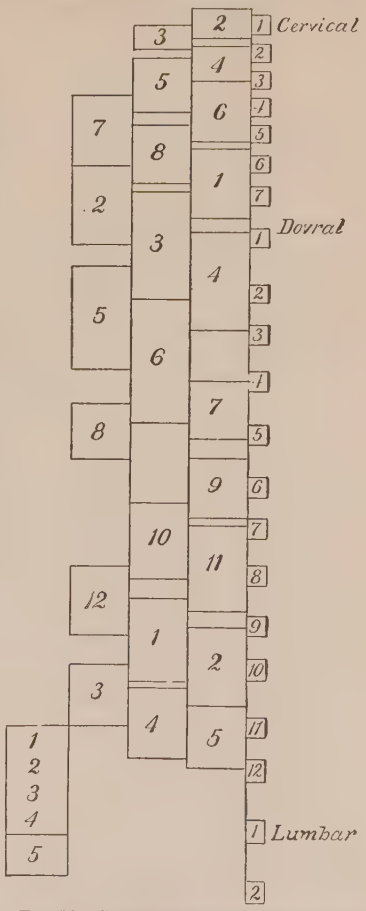


Fig. 72.—Showing the Relation of the Spinous Process to the Roots of Origin of the Spinal Nerves. The small squares to the right indicate the spinous processes; the large spaces indicate the nerve roots and the variation vertically in their origin. The relation of nerve root to spine, therefore, varies considerably (Reid).

Treatment.—General constitutional treatment consists in the use of anti-rheumatics, such as the salicylates. The muriate of ammonia has done me good service. In women pelvic troubles should be looked for; in both sexes the eyes must be attended to. Locally, counter-irritants, cupping, and leeching are useful; mustard and capsicum pastes are often a great relief. The ice-bag also is of service. Trephining the occipital bone has cured one obstinate case, and resecting the occipital nerves another.

Group II. The Lower Cervical Nerves and Brachial Plexus.

Anatomy and Physiology.—The anterior branches of the lower four cervical nerves and first dorsal nerve unite to form the brachial plexus. This gives off *short nerves* to the shoulder and trunk and *long nerves* to the arm.

The mode of formation of the brachial plexus is shown in the diagram (Fig. 73). It is in accordance with the descriptions of Walsh and Allen. The *short* or upper branches supply the shoulder and intercostal muscles. The *long* or lower branches supply the arm and hand. The neurologist needs to know: (1) the muscular distribution of each nerve and the function of the muscle; (2) the cutaneous sensory distribution; and (3) the level of origin of the nerves.

The accompanying figures and table give these points, and will be found useful for study and reference. They are based upon the investigations of Ferrier and Yeo, Thorburn, and also on Abbe's and my own experiments.

The Arrangement of the Brachial Plexus.—It is made up of three nerve trunks, which in turn make up three cords, these cords giving off various branches, thus:

1. Trunk from sixth and seventh cervical roots → forms outer cord, which gives off → Ext. ant. thoracic; Musculo-cutaneous; Outer head median.
2. Trunk from eighth cervical and first dorsal roots → forms inner cord, which gives off → Inner head median; Ulnar; Int. cutan.; Int. ant. thorac.; Intercost.-hum.
3. Trunk from fifth, sixth, seventh, and eighth cerv. and first dorsal → forms posterior cord, which gives off → Subscapular; Circumflex; Musculo-spiral.

The following table shows the origin, muscular distribution, and effect of paralysis on the motor but not on the sensory nerves. This latter is indicated in the figure (Fig. 71):

Nerves and Roots of Origin.	Muscular Distribution.	Function as Shown by Effect of Paralysis.
Posterior thoracic. Fifth and sixth cervical.	Serratus magnus.	Posterior edge of scapula is rotated out when arm is raised and carried forward. Weakening of elevation of shoulder and of inspiration.
Circumflex. Fifth, sixth, seventh, and eighth cervical.	Deltoid. Teres minor.	Loss of power to raise arm.
Suprascapular.	Supraspinatus.	Weakened power to raise arm; head of humerus tends to fall. Loss of abductors, motion forward and inward rotation of humerus.
	Infraspinatus and teres minor.	Loss of rotation of humerus outward.
Subscapular, short. Fifth and sixth cervical.	Subscapulares. Teres major.	Weakens inward rotation of arm. Weakens power of elevating shoulder.
Subscapular, long. Fifth, sixth, seventh, and eighth cervical.	Latiss. dorsi.	Weakens power to depress shoulder and to pull arm backward and to side.
Anterior thoracic.	Pectoralis major.	Loss to pull arm down and forward and to shrug the shoulder.
Musculo-cutaneous. Fourth and fifth cervical.	Biceps and brachialis anticus.	Loss of flexion of forearm and weakness of inspiration.
Musculo-spiral. Fourth, fifth, sixth, seventh, and eighth cervical.	Triceps. Supinatores.	Loss of extension of forearm. Loss of supination.
	Extensor carp. rad. Extensor carp. uln.	Extension of wrist lost except when fingers are flexed; lateral deviation.
	Extensor comm. digit. Extensor p. i. pollic. Extensor s. i. pollic. Extensor oss. met. pollic.	Impaired extension of first phalanges and wrist. Impairment of extension of thumb.
Median. Fifth, sixth, seventh, and eighth cervical.	Pronatores. Flexor carp. rad. Flexor sublim. dig. Flexor profund. dig. radial half. Two lumbricales. Abductor pollic. Flexor pollic.	Loss of pronation. Weakened flexion of wrist. Weakened flexion of second and third phalanges of first and second fingers. Loss of abduction and flexion of thumb.
Ulnar. Eighth cervical, first dorsal.	Flexor carp. ulnar. Flexor profund. dig. ulnar half. Interossei. Two lumbricales. Flexor minor digit. Adductor pollicis. Inner half of flexor brev. pollicis.	Weakened flexion of wrist; radial deviation. Weakened flexion of second and third phalanges of third and fourth fingers. Loss of flexion of first phalanges and of extension of second and third.

DISEASES OF THE LOWER CERVICAL NERVE GROUP AND THE BRACHIAL PLEXUS.—The nerves of this group are subject to the pathological disturbances common to all nerves. I shall describe them from the clinical side, which gives the following disorders:

Paralyses.
- Combined arm palsies.
- Upper-arm type or Erb's palsy.
- Lower-arm type.
- Paralysis of individual nerves.

Spasmodic Disorders.	Occupation neuroses.
Sensory Disorders.	Brachial neuralgia. Digital neuralgia. Numb-hands.
Secretory, Trophic, and Vasomotor Disorders.	Neurotic œdema.

Brachial Paralyses, Arm Palsies.—These occur as combined or total arm palsies, upper-arm and lower-arm types, and paralyses of single nerves.

Combined paralysis of the brachial nerves is a condition in which all or nearly all the branches of the plexus and its branches are involved. Total arm palsies make up about six per cent of all peripheral paralyses, and are about one-fifth as frequent as single-nerve paralyses.

Etiology.—They occur oftenest in men, but are not rare in infants, being then due to injuries during parturition. After infancy they are most frequent in early and middle life.

The exciting causes are obstetrical and other injuries; deep-seated inflammations and tumors; shoulder dislocations; primary neuritis; crutch and other forms of mechanical compression; functional palsies, from overwork and hysteria; in rare cases, spinal-cord and brain disease.

The *symptoms* vary with the severity and extent of the lesion.

With regard to severity, there are three degrees. In the first there is simply a transient palsy, due to lying too long on the arm. The arm feels heavy, numb, and "asleep." In a few minutes or hours this palsy disappears. In the second degree the nerves are so much compressed as to be mechanically injured. If the patient has been drinking hard, even moderate pressure may set up an inflammatory or destructive process that leads to quite a serious palsy. In the third degree the nerves are actually cut or torn across, or so compressed as to lose their anatomical integrity.

The resulting symptoms are those common to all nerve injuries, viz., paralysis, wasting, changes in electrical reaction of the muscles. Pain, tenderness, anæsthesia, trophic, secretory, and vasomotor disturbances are also present in varying degree.

The distribution of the paralysis, atrophy, and sensory disturbance depends, of course, upon the arm nerves chiefly involved. The diagram (Fig. 71) will enable one to see in any case where the trouble is localized.

There are three common symptoms, however, of which it is often very important to analyze the cause. These are the loss of

power for elevation of the arm and for flexion and extension of the forearm.

Flexion of the forearm is performed by the biceps and brachialis anticus, and is helped by the supinator longus. These muscles are supplied by the musculo-cutaneous nerve, except the supinator, which is supplied by the musculo-spiral. Hence when a person cannot flex the forearm, the musculo-cutaneous is chiefly affected.

Extension of the arm is done by the triceps, which is supplied by the musculo-spiral.

Elevation of the Arm Outward.—Inability to raise the arm is the common and striking symptom in combined brachial palsies. The arm is raised by a number of muscles. The deltoid acts first and most, but it can only raise the arm to a right angle. It is supplied by the circumflex nerve from the posterior cord of the plexus. After the arm is raised to a right angle, it is further elevated by rotating the scapula, and this is done chiefly by the middle part of the trapezius (lower cervical and upper dorsal nerves) and by the serratus magnus, supplied by the posterior thoracic nerve. A number of other muscles combine to strengthen elevation of the shoulder, but this action can only be abolished by paralysis of the deltoid or trapezius and serratus magnus.

The *diagnosis* of these cases involves, first, the consideration of the seat of the lesion and the special nerves involved; next, that of the nature of the lesion. A recognition of the seat of the lesion and of the special nerves involved depends entirely upon the study of the distribution of the palsy and of the atrophy and sensory disturbance. It is important to determine whether one is dealing with a total arm palsy, an upper-arm type (Erb's palsy), or a lower-arm type.

In *Erb's palsy* there is involvement of the deltoid, biceps, brachialis anticus, and supinator longus, with at times paralysis of the supinator brevis, and even of all the muscles supplied by the median nerve. The lesion is either in the cord formed by the fifth and sixth cervical nerves or a little lower in the brachial plexus, where the fibres supplying the musculo-spiral, circumflex, and musculo-cutaneous lie close together. At all events, the lesion involves the central parts and upper cords of the plexus. The arm hangs by the side and the forearm cannot be flexed.

In the *lower-arm type* the triceps, the flexors of the wrist, the pronators, the flexors and extensors of the fingers, and the hand muscles are involved. The arm can be raised and the forearm flexed and supinated, but the hand is useless and the extension of the forearm is impossible. The lesion here involves chiefly the nerves from the seventh and eighth cervical and first dorsal roots.

If the lesion is in the nerve there will be atrophy, changes in electrical reaction, sensory disturbances, and often, if there is neuritis, pain over the nerves. The reflexes will be lessened or absent. If the lesion is in the spinal cord, symptoms in other parts of the body will be present, or, if not, there will be no sensory disturbance, as in an arm palsy from anterior poliomyelitis. In rare cases arm palsies may be caused by spinal tumors or a local meningitis, in which case diagnosis is difficult.

The *prognosis* is usually good. Nearly all these cases get well, the duration of the incapacity being from two or three months to a year. Even in the severest cases recovery is possible after one or two years. If, however, the nerves are torn across and the ends

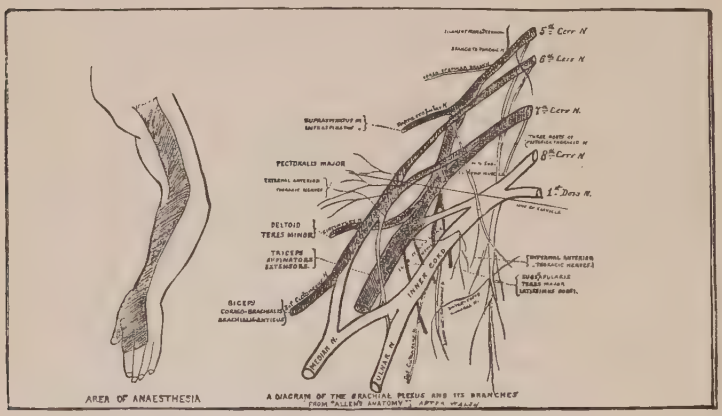


Fig. 73.—Illustrating the Formation of the Brachial Plexus; also the Involvement of the Plexus in Degenerative Neuritis (Leszynsky).

widely separated, recovery is doubtful unless an operation is promptly done.

The *treatment* consists in electrical applications, mechanical support, with potassium iodide internally and abstinence from alcohol. Local injections of nitrate of strychnine are useful, and massage should be used if it can be applied carefully.

In brachial palsies due to severe injuries, dislocations, fractures, etc., in which there is evidence, from the extreme atrophy and absence of electrical reaction, that the nerve is entirely cut across and that the ends are not in apposition, a surgical operation is stringently needed. The nerves should be exposed and the ends brought as near together as possible. Decalcified bone-tubes or sterilized macaroni may be used to give a passage for the central end to grow into the peripheral. In these cases, however, it must be remembered that the two ends do not unite; but the

central end grows down into the tract of the old degenerated peripheral stem.

The *upper-arm type* and *lower-arm type* palsies are caused by much the same factors as the combined palsies; their symptoms have been indicated above. The upper-arm type is especially frequent in infants and constitutes one of the obstetrical paralyses. It may in some cases be due to injury or hemorrhage in the cord.

A peculiar form of combined nerve palsy sometimes occurs, due apparently to a *primary brachial neuritis*. It begins in the plexus and involves first the nerves of the upper cervical roots. It may extend down and involve the ulnar, median, or musculospiral.

It occurs in adult men generally and in those exposed to rheumatic influences. Perhaps lead-poisoning may exist.

It begins gradually with slight pains and weakness in the shoulder and arm muscles. Atrophy and anæsthesia follow, and degeneration reactions are present. There is not a great deal of pain (Fig. 73). The disease is usually confined to one side. It lasts several months, and nearly complete recovery finally takes place. Relapses may occur.

It is differentiated from progressive muscular atrophy by the pain, anæsthesia, and electrical reactions and from arthritic atrophy, by the absence of any history of arthritis and the presence of degeneration reactions and anæsthetic areas.

Paralyses of Single Nerves.—Paralysis of the Posterior Thoracic Nerve.—*Etiology.*—This rare trouble usually occurs in male adults and is due to injury or sudden strains. Its paralysis may be part of a progressive muscular atrophy. The nerve goes to the serratus magnus.

Symptoms.—When paralyzed, there is difficulty in raising the arm above the horizontal position and the movements of the shoulder are weakened. When the arm hangs by the side the lower angle of the scapula is drawn a little nearer the vertebral column and protrudes slightly. When the arm is held out horizontally the inner edge of the scapula protrudes and is drawn toward the middle line. When the raised arm is brought forward there is a deep groove formed between the inner border of the scapula and the thoracic wall.

The disease often runs a long course and is accompanied with pain.

Paralysis of the Circumflex Nerve.—The nerve goes to the deltoid, teres minor, third head of the triceps, and shoulder joint. It gives sensation to the skin of the shoulder. It is very often paralyzed. The commonest cause is a fall or injury, dislocation, and rheumatic inflammation of the joint. The arm can-

not be elevated or rotated outward. There is atrophy, anæsthesia, and sometimes pain.

Paralysis of the Suprascapular Nerves.—The nerve goes to the spinati muscles, teres minor, and shoulder joint. Disease of this nerve alone is rare.

The supraspinatus rotates the shoulder in, the infraspinatus and teres minor rotate it out. When paralyzed, there is an impairment of rotation and some impairment of elevation of the shoulder.

Paralysis of the Musculo-Spiral Nerve (*Wrist Drop, Lead Palsy, Compression Paralysis*).—The distribution of this nerve is given in the table and cut. Its function is to extend and supinate the forearm, to extend the wrist and fingers, and to adduct and abduct the fingers slightly. It extends directly only the last or ungual phalanges, the first and second phalanges being extended by the ulnar nerve.

Etiology.—The musculo-spiral, owing partly to its course, is the most frequently affected by paralysis of all the arm nerves. Pressure on the nerve during sleep, especially when the patient is intoxicated, crutch-pressure, fractures, wounds, tumors, lead-poisoning, arsenical, alcoholic, and other forms of multiple neuritis are the causes of its disordered function.

Symptoms.—The symptoms of this paralysis are "wrist drop," due to an inability to extend the wrist or fingers. The first and second phalanges can be extended somewhat by the interossei and lumbricales, but the last phalanges cannot be extended at all. The first finger is least affected. The fingers can only be slightly abducted, supination is generally lessened or lost; if the lesion is high up, the triceps is involved and the power of extending the forearm weakened. There may be atrophy of the muscles and degeneration reaction. A swelling over the tendons of the wrist joint may occur. Some numbness and tingling exists, and occasionally there is anæsthesia in the distribution of the radial nerve on the hand. The disease lasts but a few weeks if due to compression; for months if due to neuritis, lead-poisoning, or severe injury of the nerve. Eventually recovery takes place.

When the disease is due to *lead-poisoning* there are some peculiarities in its course. Thus the supinator longus usually escapes; the palsy begins gradually and usually involves both arms; it may extend to the upper arm. Partial degeneration reactions are present. There is rarely any anæsthesia and but little pain. There is a lead line on the gum and a history of constipation and colic.

In *alcoholic* and other forms of multiple neuritis there is pain and paræsthesia, both arms are involved, and the flexors and

other forearm muscles are somewhat implicated. There are marked sensory disturbances. The legs are also affected.

In *compression palsy* the supinators and often the triceps are involved.

When the lesion of the nerve is high up, as in *crutch paralysis*, there is but little anæsthesia, and that is found on the anterior surface of the forearm, in the distribution of the external and internal cutaneous nerves. Lesion of the nerve lower down may give rise to some anæsthesia along the radial border of the forearm and back of hand; but the anæsthetic area varies a great deal.

The *diagnosis* of the paralysis is easily made. The most important point is to find out the cause. The different characteristics of lead palsy, neuritic palsy, and compression palsy have been indicated in the description of the symptoms. One must be sure to exclude also progressive muscular atrophy.

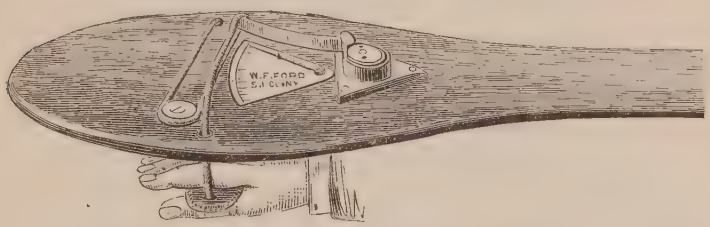


FIG. 74.—AUTHOR'S DYNAMOMETER FOR MEASURING THE STRENGTH OF EXTENSOR AND FLEXOR MUSCLES OF THE FOREARM AND HAND.

The *treatment* consists of mechanical measures such as electricity, massage, the application of rubber muscles, and in bad cases the fixation of the forearm and hand in hyperextension by means of a splint and plaster-of-Paris bandage (Gibney). Internally in the early stage it is best to administer iodide of potassium and sulphate of magnesium (in lead palsy), the salicylates or nitrate of silver in neuritis; later, hypodermic injections of strychnia may be given. Static sparks, galvanism, and other forms of electricity unquestionably do good in some cases, as I have had opportunity to prove.

PARALYSIS OF THE MEDIAN NERVE is rare as an isolated trouble, and is almost always due to injury or neighboring lesions. Embolism of the axillary artery after labor has produced it.

When paralysis occurs the grip is weakened. Flexion and abduction of the thumb and flexion of the first and second fingers are impaired. Atrophy of the thenar eminence may occur. The anæsthetic area varies, but is well shown in the accompanying cut from Ruxton (Fig. 75).

Paralysis of the Ulnar Nerve.—*Etiology.*—The ulnar nerve is rather commonly affected by paralysis, the occurrence ranking next in frequency to musculo-spiral palsy. It is rarely affected in lead-poisoning, but is usually early involved in progressive muscular atrophy. It is sometimes attacked by a primary degenerative neuritis. Injuries are the common cause.

The *symptoms* are shown by the table. The hand cannot be closed tightly, the little and ring fingers being especially weak. The first phalanges are drawn back and the second and third phalanges flexed; when the interossei and lumbricales atrophy, the result is the "griffin claw" or *main en griffe*. The fingers cannot be adducted or abducted except feebly.

There is anæsthesia over the area of distribution of the ulnar (Fig. 75); there may be pain and tenderness.

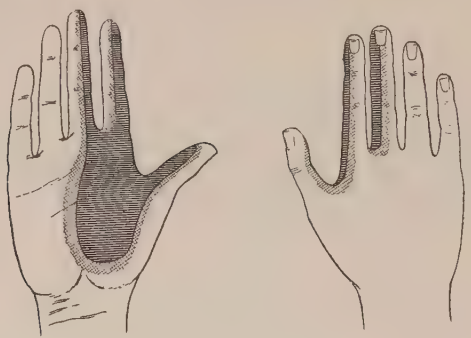


Fig. 75.—Area of Anæsthesia in Total Paralysis of the Median Nerve.

Symmetrical Spontaneous Ulnar Neuritis.—A form of neuritis of the ulnar nerve sometimes occurs which has certain peculiar characters. It develops slowly in persons with a neuropathic history, and without known exciting cause. There is first some pain and paræsthesia in the region of the ulnar nerve of one hand; this is followed by weakness and atrophy of the muscles supplied by the ulnar nerve, and characteristic deformity appears. Anæsthesia develops coincidently. The other hand soon becomes affected.

The disease is very chronic, and complete recovery is rare.

It is probably a form of degenerative neuritis.

Spasmodic Disorders of the Arm.—The arms and hands are especially subject to tremors, choreic and other spasmodic movements. The only spasmodic disorders, however, which may be said to be especially located there are writer's cramp and allied occupation neuroses. These are described elsewhere.

Sensory Disorders of the Brachial Nerves.—Cervico-Brachial Neuralgia.—This is a painful affection involving

the sensory nerve of the neck, shoulder, and arm. It is a somewhat rare form of neuralgia, and like most other forms occurs oftenest in women, before middle life. The ordinary causes of neuralgia produce it, but rheumatism and gout are rather prominent factors. Reflex irritation from carious teeth and uterine disease have been found to be causes. It occurs symptomatically in locomotor ataxia and other cord diseases. A low grade of neuritis probably often exists.

Symptoms.—It begins somewhat gradually with aching pains in the neck, shoulder, axilla, and along the course of the nerves. The pains gradually increase and are usually worse at night. The pain is increased by use of the arm and by exposure. Only one arm is affected. Painful points may be felt in the axilla, at the lower end of the scapular, over the deltoid, over the ulnar near the wrist, over the lower part of the radial and sometimes on each side of the lower cervical vertebræ. There are usually paræsthesiæ and numb feelings. If there is a complicating neuritis, burning sensations are felt ("causalgia"). There may be anæsthesia, vasomotor disturbances, herpes, and muscular weakness and atrophy. Brachial neuralgia generally involves all the nerves of the plexus. If special nerves are involved the ulnar is oftenest affected, next the musculo-spiral, and last the median.

Digital neuralgia sometimes occurs. The pain is often located in a single finger. The cause is usually a local injury or neuritis. Sometimes it is a reflex pain due to some remote trouble. In a few cases this remote trouble is uterine.

Diagnosis.—The etiological diagnosis is most important. Inquiries as to a rheumatic or gouty element should be made. The presence of any serious amount of neuritis would be shown by local tenderness and motor weakness. Organic cord disease must be excluded.

The *prognosis*, if the neuralgia is functional, is good. If a neuritis complicates it, it is more serious. If the neuritis, however, is slight, and is secondary to a trauma, or is rheumatic or gouty, the prognosis is favorable. Ordinary attacks last about six weeks.

The Treatment.—Salicylate of potassium, iodide of potassium, or muriate of ammonia should be given and in large doses. Aconitia and gelsemium are of not much value. The electrical current, either faradic or galvanic, has a very decided effect for good.

Leeching helps some cases. Blisters may be used in neuritic cases. The analgesic drugs like phenacetin and antipyrine and antifebrin are useful symptomatically. Hot local applications often give some relief. Colchicum cures some cases of gouty origin.

Migrating neuritis is a serious but rare malady which deserves some mention. It occurs as a sequel to some wound of or operation upon a nerve. The local neuritis extends usually up the arm (ascending neuritis). It is accompanied with intense pain, anæsthesia, paralysis, and atrophy.

The disease is very chronic and intractable.

Group III. The Dorsal Nerves.

Anatomy and Physiology.—The dorsal nerves are twelve in number. The first is the largest and belongs functionally to the arm nerves. The dorsal nerves carry motor and sensory fibres to the voluntary muscles, skin, and other tissues of the trunk wall. They also carry splanchnic fibres to the lungs and abdominal viscera. They divide into anterior and posterior branches. The anterior form the *intercostal nerves*, of which the first six are distributed to the wall of the thorax and the last six to the wall of the abdomen. All these nerves give off lateral and anterior branches. The posterior branches of the dorsal nerves are small, and supply the muscles and skin of the back.

Besides the above, there are recurrent branches which enter the spinal canal and supply the intraspinal veins and membranes. The first six dorsal nerves contain fibres which enter the sympathetic and supply the pleura and lungs. Fibres from the last six enter the sympathetic and supply the pelvic viscera. The intercostal nerves in their course lie part of the way beneath the pleura; they also pass along in close company with the intercostal artery and vein, lying just above these in the groove on the internal surface and inferior border of the ribs.

The upper six dorsal nerves, including both branches, are mainly inspiratory in function. They also extend and rotate the dorsal and cervical vertebræ. The lower dorsal are expiratory nerves; they also assist in compressing the abdominal viscera, and in flexing, extending, and rotating the spine.

The diseases of the intercostal nerves are almost entirely of a sensory character. They are intercostal neuralgia, symptomatic side pains, herpes zoster. Paralyses and anæsthesiæ occur in connection with vertebral and spinal-cord diseases.

Intercostal Neuralgia; Side Pains.—*Etiology.*—This is a very common neuralgia. It occurs much oftener in women (partly from corset-pressure) than in men (seven to one). The favorite age is twenty to thirty-five; the season, winter. Anæmia, neurasthenic and hysterical conditions, child-bearing, pelvic disorder, dyspepsia, heart disease, malaria, and lead-poisoning are frequent causes. Exposure and muscular strain are rare exciting causes.

Symptoms.—The disease comes on suddenly. The pain in typical cases is sharp and stabbing, but not much increased by respiratory movements. There are tender points at the seat of pain, which is usually greatest over the side at the exit of the lateral nerve branches. Often a tender point is felt over the exit

of the dorsal or of the anterior branch. It is rare to find all three points. The disease runs about the course of facial neuralgias, *i.e.*, of two to six weeks, but it is sometimes very obstinate, lasting for many months. The sixth to tenth nerves are those oftenest involved. The left side is more susceptible than the right (three to one).

Pathology.—In most cases the nerve is irritated by poor or poisoned blood. In a minority of cases the pain is reflex from stomach, pelvis, or heart. Sometimes there is a neuritis.

Diagnosis.—Probably one-half the pains in the side are myalgic in nature, and should be classed as pleurodynia. These pains can be distinguished by the history of their origin and of rheumatic influences, by their diffuseness and dulness, by the great tenderness on pressure, and the pain produced on taking a deep breath.

There is another considerable proportion of cases in which the pains are mainly neuralgic, but yet there are some evidences of muscular complications. Some of these pains are reflex.

In the third class of cases there is the pure stabbing neuralgia. The diagnosis is based on the exclusion of pleurisy, rheumatic and reflex causes, by the character of the pain, and the presence of tender points.

Prognosis.—The prognosis is good, except for a few chronic cases due probably to a degenerative neuritis. In some of these cases there is lead-poisoning.

Treatment.—The most efficacious treatment is a blister and ferruginous tonics with quinine. In all cases the heart, pleura, stomach, and pelvic organs must be examined, and any disorder relieved. Iodide of potassium and chlorodyne have cured some very bad cases in my experience. If there is a rheumatic and muscular element, give salicylates or the analgesics, apply heat, and secure rest by adhesive straps.

MAMMARY NEURALGIA (MASTODYNIA) is a form of intercostal neuralgia involving the anterior and lateral branches of the three or four upper dorsal nerves.

Etiology.—It is caused by local tumors, or it may be an essential neuralgia. The causes in the latter class are anæmia, pendent breasts, pressure from badly fitting corsets, and injury.

Mammary neuralgia also occurs in hysterical women and young girls sexually precocious; it may occur in pregnancy and during lactation. Many mammary pains are due simply to local disorder of the gland.

Symptoms.—Mammary neuralgia is unilateral, often very severe, and if it occurs in middle life is liable to cause much mental depression from fear of cancer.

The *treatment* depends upon the cause. It requires general

tonic measures and attention to the proper support and protection of the gland.

HERPES ZOSTER. DERMATITIC NEURITIS (*Shingles*).—Almost the only recognised form of neuritis of the intercostal nerves is known under the name of *herpes zoster*. This is an acute dermatitis, secondary to the neuritis.

Etiology.—Its causes are wounds, infections, the morphine habit, rheumatic, gouty, and syphilitic poisons, and emotional influences. The inflammation affects not only the nerves, but the spinal ganglia.

Symptoms.—It begins gradually with the development of pain and a herpetic eruption upon one side of the trunk. It generally involves the lower dorsal nerves. The eruption follows the course of the nerve, rarely extending to the opposite side. The pain gradually subsides, and the disease itself runs its course in a few weeks.

Treatment.—In the early stage the galvanic current is efficacious. Local anodynes and protective ointments may be applied. Anti-rheumatics and analgesics are to be administered internally.

GROUP IV. THE LUMBAR NERVES.

Anatomy and Physiology.—The lumbar nerves are five in number. The posterior branches supply the erector spinæ, interossei, multifidi spinæ and interspinales, and also the skin of the back. The anterior branches of the upper four unite to form the *lumbar plexus*. The fifth or lumbo-sacral nerve sends most of its fibres to the sacral plexus. The branches of the lumbar plexus are: (1) the ilio-inguinal, (2) ilio-hypogastric (from first lumbar), (3) genito-crural and (4) external cutaneous (mainly from the second), (5) obturator (from third and fourth lumbar), (6) the anterior crural (from second, third, and fourth—Fig. 70).

The first four branches of the plexus are comparatively short and supply sensation to the abdominal wall and external genitals. The last two are longer and supply the hip and knee joints, the muscles of the anterior, inner, and outer part of the thigh, the skin over this region and the inner side of the leg and dorsum of the foot (Fig. 76).

The diseases of the lumbar nerves and plexus so far as they form independent disorders are mainly the neuralgias.

PARALYSES AND SPASMODIC TROUBLES OF THE LUMBAR NERVES are not rare, but are usually symptomatic of some extrinsic and often serious disease.

Etiology.—Hence it is well to catalogue here those affections which may produce lumbar palsies or spasms. They are pelvic tumors or injuries, impacted feces, caries of the spine, psoas abscess, obturator hernia, hip disease, and pressure of the fœtal head.

Symptoms.—When the upper two lumbar nerves are involved, only sensory symptoms in the distribution of their branches occur (Fig. 76). If the next two are also involved, there may be trouble in extending the leg and flexing the hip on the trunk. The patient cannot raise himself to a sitting posture. If there is irritation and spasm, the thigh is drawn up and adducted.

In making a diagnosis of lumbar-nerve disease, one should remember that of the six branches of the plexus the upper four are mainly sensory, the lower two mixed nerves.

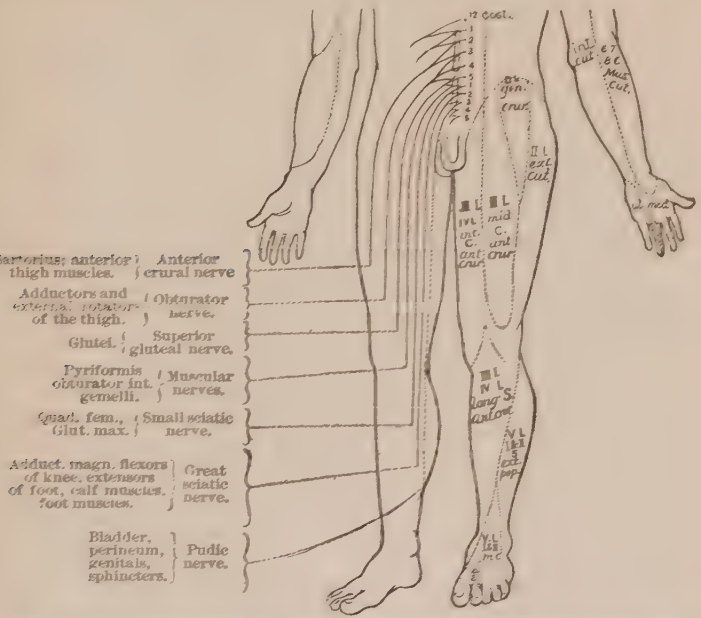


Fig. 76.—Diagram showing the Origin and Distribution of the Motor and Sensory Nerves of the Lower Limbs.

In *paralysis of the obturator nerve* there is loss of power to adduct the thigh and cross the leg, and weakness of outward rotation of the thigh. Anæsthesia over the inner side of the thigh may be present.

In *paralysis of the anterior crural nerve* there is weakness of the muscles of the anterior surface of the thigh, loss of power of extending the leg, and anæsthesia or pain over the crural area.

Paralysis of the posterior branches of the lumbar nerves causes weakness or paralysis of the erectors of the spine. The lumbar curve is very greatly exaggerated, the shoulders being

thrown far back and the belly protruding. This condition occurs in progressive muscular atrophy, particularly in the pseudo-hypertrophic form.

Neuralgias and Reflex Pains of the Lumbar Nerves. —*Etiology.*—Neuralgias of the upper branches of the lumbar nerves are called lumbo-abdominal. They occur oftener in women and usually after the thirtieth year. To the ordinary causes of neuralgia we add straining, constipation, and pelvic disease. True essential neuralgia is rare; but myalgic and reflex pains from uterine disease are very common, especially in women.

Symptoms.—There is pain in the loins, back, and buttocks, extending down to the hypogastrium or genitals on one side. The pain in the back, however, is often bilateral. Painful points may be found after a time, as in intercostal neuralgia. Sometimes the pain is located in the side of the penis (penile neuralgia). Neuralgia of the long lumbar branches is called femoral or crural. A common form of this constitutes what is called *painful thigh.* The patient complains of pain in the front of the knee and the anterior and outer parts of the thigh, but has no pain posteriorly and none below the knee. The internal branches of the anterior crural nerve do not seem to be affected, while the middle and external cutaneous branches and the genito-crural nerve are involved.

Reflex and Symptomatic Pains.—Disease of the hip or of the sacro-iliac joint or vertebræ may cause a reflex pain in the obturator nerve, localized especially in the knee and back of this joint. The fœtal head sometimes compresses these branches, causing a symptomatic neuralgia. Diseases of the internal genital organs are especially liable to cause reflex pain in the lumbar nerves. Diseases of the external genitals and bladder more often reflect pains upon the sacral nerves. In biliary colic pains are felt in the ilio-inguinal and hypogastric nerves. Local disease of the psoas muscle or iliacus, in the neighborhood of Poupart's ligament, causes pains in the lumbar nerves. Lumbar neuralgias generally run a favorable and not very long course.

Diagnosis.—They are distinguished from lumbago by the unilateral position, the distribution and paroxysmal character of the pain, and the lack of severe suffering on motion and pressure; the tender points and the absence of any organic disease.

Lumbago comes on suddenly, with a history of exposure, is confined to a single group of muscles, which are tender on deep pressure. In lumbar sprain the onset is also sudden, with a history of injury, great local tenderness, and evidences of trauma.

The *treatment* is the same as that for neuralgia in general. The frequent presence of pelvic disease and of anæmia and a rheumatic history must be borne in mind.

Group V. The Sacral Nerves.

Anatomy and Physiology.—The sacral nerves are five in number. The first four divide into anterior and posterior branches. The fifth has no anterior branch. The posterior branches escape through the posterior sacral foramina and supply the multifidus spinæ and the skin over the sacrum and coccyx.

The first three anterior branches, with the lumbo-sacral nerve and a branch from the fourth sacral, unite to form the sacral plexus. This lies upon the pyriformis muscle in the pelvis, and escapes at the lower part of the sacro-sciatic foramen. The great mass of the fibres go to make up the sciatic nerve.

The roots of origin of the sacral and coccygeal nerves form the canda equina.

The branches of the sacral plexus are the superior gluteal, muscular, small sciatic, inferior gluteal, pudic, great sciatic, perforating, cutaneous, and articular. These are distributed to the muscles, skin, and joints of the buttocks, thighs, legs, and feet. Their distribution is shown in Fig. 76.

The sacral nerves are the main agents in station and locomotion. They control the legs entirely, also the posterior muscles of the thigh and buttocks; they give sensation to these parts. They carry also fibres that regulate the sexual function, bladder, and rectum. From the sacral portion of the cord there is an outflow of nerves to the sympathetic, thence to the pelvic organs.

The diseases of the sacral nerves may be classified in a similar way to those of the brachial plexus. I shall here also follow the clinical method and study the paralyses, spasmodic disorders, and neuralgias of this group of nerves.

Peripheral Leg Palsies.—Paralyses of the lower limbs from disease of the nerves may be either combined or single, just as is the case with arm palsies.

A combined sacral palsy is one in which all or nearly all of the branches of the sacral plexus are involved.

Etiology.—Such palsies are due to much the same causes as those affecting the lumbar nerves, viz., injury, dislocation, hip disease, tumors, and neuritis. Hysteria may cause a functional sacral palsy.

Symptoms.—The symptoms are indicated by a study of the distribution of the nerves, varying, however, in degree. The foot cannot be moved; the leg can be slightly flexed by the anterior crural, but not extended; the thigh cannot be drawn back freely or rotated perfectly. There is anæsthesia over the distribution of the sacral nerves; pain may be present; wasting and vasomotor and secretory disturbances occur unless the paralysis is functional.

The course depends on the severity of the lesion. If the nerve is totally cut or torn across, it may require one or two years for perfect recovery, which, however, occurs if the severed ends are properly approximated.

The *diagnosis* of a sacral palsy is based on the history and on the distribution of the anæsthesia, and of the muscular paralysis. The sacral nerves do everything for the lower limb except extend the leg, flex and adduct the thigh, and to some extent rotate it. They supply sensation equally extensively (see Fig. 76).

The distinction from spinal-cord disease is chiefly based on the unilateral symptoms, the absence of disorder of the sphincters, and the combination of paralysis, wasting and sensory troubles, in the course of the sacral nerve.

Single-Nerve Sacral Palsies.—The symptoms of paralysis of single nerves are indicated by their function. The nerves rarely affected are the superior gluteal, muscular, and small sciatic. The nerve oftenest affected is the *great sciatic*, and especially its *anterior tibial branch*. In the latter case a condition called "drop foot" is produced.

In the *pathology* and *treatment* of sacral palsies there is nothing especial that can be said.

Spasmodic Disorders of the Sacral Nerves.—Tremor, rigidity, clonic and tonic spasms, myoclonus, athetoid movements, all affect the lower extremities, but they are almost invariably part of some general or central disorder, such as chorea, paralysis agitans, hysteria, etc. Under the head of occupation neuroses there occur certain rare spasmodic troubles special to the legs. Saltatory spasm involves the legs alone. These disorders are, however, general neuroses.

Sciatica (Neuralgia of the Sciatic Nerve, Sciatic Neuritis).—This is a form of neuralgia occurring in middle life and characterized by intense pain in the course of the sciatic nerve. A large proportion of the cases are due to a neuritis.

Etiology.—The disease occurs three times as often in men as in women, and is the only neuralgia of which this can be said. Most cases in this country occur between the ages of forty and fifty; next between thirty and forty.*

The gouty and arthritic diathesis, occupations which lead to exposure and strain, predispose to the disease. It is not rare, therefore, among laboring men. In younger persons a neurotic constitution predisposes to the disease, and in this class the trouble is more truly of a neuralgic character and less of a neuritis.

Most cases occur in the autumn and winter. The exciting causes are constipation, pressure from hard seats, exposure, muscular strain from heavy work, and pelvic disorders. Symptomatic

* Personal statistics (102 cases) and those of Dr. L. Putzel (53 cases) give males. 111; females, 44. Ages: 10-20, 4; 21-30, 30; 31-40, 43; 41-50, 44; 51-60, 18; 61-70, 12; 71-80, 4.

sciatica may be caused by the pressure of pelvic tumors, injury to the nerves, inflammation, vertebral and spinal disease; sciatica occurs in diabetes and in phthisis. In elderly persons of a rheumatic constitution inflammatory processes about the hip joint complicate or cause the neuralgia.

Symptoms.—The disease begins rather suddenly. Pain is felt in the back of the thigh, running down the leg in the course of the nerve. Generally it is most marked in the thigh, extending up often into the lumbar region. Sometimes the disease begins like a lumbago; more rarely pain is first felt in the calf or foot. The pain is increased by motion, and the patient holds himself in a constrained position. The pelvis is tilted up toward the sound side and the trunk inclined over to the diseased side. After a time this leads in some cases to a characteristic deformity (sciatic scoliosis) in which the convexity of the curve of the vertebral spines is directed toward the diseased side. The pain is almost continuous, with paroxysms of great severity, which often occur at night. During these paroxysms the pain is sharp, burning, and lancinating. In the intervals it is dull. Besides the pains the patient suffers from feelings of numbness, tingling, and a sense of coldness and weight in the affected limb. There are almost always tender points over the course of the nerve. These may be found at the sciatic notch, at the middle of the hip, behind the knee, just below the head of the fibula in the middle of the calf, behind the external malleolus, and on the back of the foot (Fig. 77).

A pain running up the back of the thigh may be caused by pressure over the back of the knee when the leg is extended at a little more than a right angle. This is diagnostic (Gowers). If the patient lies on his back and the leg is kept extended, and then the whole limb brought slowly up until it is at an acute angle with the trunk, a sharp pain in the sciatic notch is felt; this too is diagnostic.

Anæsthesia over the course of the nerves occurs very rarely. When present, it indicates a severe neuritis or injury to the nerve.

Muscular wasting and weakness occur after a time, and in old and severe cases partial electrical degeneration reactions may be observed. Herpetic eruptions over the course of the nerve occur in rare cases. The affected limb usually feels colder and shows evidence of enfeebled vasomotor supply.

The disease usually lasts two or three months; not rarely it lasts six months or even a year or more. It has been known to extend slowly upward and involve the sacral plexus or even the spinal cord.

Pathology.—The trouble is, as already stated, a chronic perineuritis in the majority of cases.

Diagnosis.—Sciatica has to be distinguished from hip-joint disease, organic disease of the cauda equina or cord, muscular pains in the hip and leg, and from pains caused by tumors. Pure sciatic neuralgia ought also to be distinguished from sciatic neuritis. A consideration of the facts already given ought not to make the diagnosis difficult.

Pure sciatic neuralgia occurs in eary life and is not accompanied with much local tenderness. There is no paralysis or wasting of the limb. Double sciatica is most always symptomatic of diabetes or organic disease. True sciatica is rarely double.

Prognosis.—Almost all cases get well in from three to six

FIG. 77.—SHOWING THE TENDER POINTS IN SCIATICA.

FIG. 78. PLANTAR FORM OF SCIATICA; PLANTAR NEURITIS, showing area of pain (Mitchell).

months. Severe attacks in people over forty are the most intractable. Relapses occur, but not as a rule.

Treatment.—In all cases which are seen early, the most important indication is rest. The patient should be put to bed, and the whole lower extremity secured in a Thomas splint extending from ankle to axilla. Ice-bags or linseed poultices or leeches should then be applied over the course of the nerve. A blue pill (gr. v.) may be given twice daily at first. In less severe or older cases large blisters should be applied over the nerve in the thigh, and the application repeated in a week. If there is a rheumatic history, potassium salicylate or iodide should be given in full doses. The bowels must be freely opened. Hypodermic injections of morphia or cocaine (gr. $\frac{1}{4}$) may be needed for a few days, the cocaine being repeated if necessary. When the disease has become more chronic a strong galvanic current may be given daily

with large electrodes, one over the lumbar region or sciatic notch; the other, which should be the positive pole, over the leg and foot. As so-called specific remedies we have oil of turpentine in doses of fifteen drops t. i. d., and this may be advantageously combined with oil of gaultheria. Massive doses of antifebrin or antipyrine sometimes stop the pains (gr. x., q. 2 h.). There are a great many local remedies which at times prove useful. Among these mustard plasters, menthol, chloroform liniment, setons, acupuncture, cups, and the actual cautery and chloride of methyl can be recommended. Bandaging the limb in sulphur to which a little menthol is added is often very efficacious. Kneading the nerve with a glass rod and an anodyne ointment is sometimes beneficial. Very little can be expected from nerve-stretching, but it may be tried as a last resort. If tried, however, great caution should be exercised in pulling on the nerve. Not over thirty to forty pounds pull should be used. The operation of cutting down on the nerve and dissecting off the sheath for a space of several inches may be tried.

Plantar Neuralgia.—In rare cases the pain of sciatic neuralgia is limited to the plantar nerves, and is accompanied with paræsthesia and even anæsthesia of this region. The condition here is probably a neuritis combined sometimes with arthritic changes. Erythromelalgia may be regarded as a form of plantar neuralgia.

Coccygodynia is a neuralgia affecting the lower posterior branches of the sacral nerves. It occurs oftenest in women and is caused by exposure, injury, and labor. Coccygeal pains occur in spinal irritation and reflexly from pelvic disease. The disease is a most annoying one, as it interferes with sitting and walking. There is often also pain at stool, and the parts are tender to pressure. The disease is usually one involving the fibrous structures of the coccyx, and is more an articular and bony than nervous disorder. Surgical treatment, such as amputation of the coccyx, may be needed.

Recapitulation.—The student should understand that, despite the great clinical variety of peripheral nervous disease, there is an underlying unity as regards the pathological condition, the cause and the symptoms. In order that this may be clearly brought out, I append the following tables.

The student will remember that the causes of nearly all peripheral nerve-diseases can be classed under the following heads:

Heredity and neuropathic constitution; *sex; age*—infant, youth, etc.; *injuries; diatheses*—rheumatism, gout, diabetes; *exposures; mental* and *physical excesses; infections*—chronic, *e.g.*, syphilis and the febrile; *poisons*—narcotic, metallic, etc.; *anæmia* and *marasmus; arterio-sclerosis.*

The table as given applies to *all* peripheral nerves. The cranial nerves are, however, more affected by syphilis and brain disease.

TABLE I.—PERIPHERAL NERVOUS DISEASES—TYPES AND CAUSES.

Type of Disease.		Principal Causes.
1. Anæmia and Hyperæmia:		
Inflammation.	Multiple neuritis.	Female, young adults. Poisons—alcohol, arsenic; Infections—febrile, endemic; Diatheses—rheumatism, Exposures.
	Mono-neuritis.	Male, infants, adults. Injuries, over-use; Poisons—lead; Infections—febrile, syphilis; Diatheses—gout, rheumatism.
Degenerations.	Primary.	Male, mid-age, senility. Marasmus, Loc. ataxia; Poisons—lead; Infections—diphtheria.
	Secondary.	Injury, destruction, or inflammation of nerve or its trophic centre.
Destruction.		Injuries, compression by tumors, etc.
Tumors.	Single.	Heredity, injuries, operations.
	Multiple.	Chronic infections.
Functional neuroses.	With signs of nerve irritation.	Heredity, neuropathy, female, youth, and mid-age. Over-use. Poisons—narcotic, metallic.
	Or of nerve depression.	Diatheses—rheumatism, gout, diabetes; Infection—syphilis; Anæmia, Arterio-sclerosis; Malnutrition.

The symptoms of peripheral nervous diseases are shown in irritation and excess, diminution and loss, and perversion of function. Since nerves are sensory, motor, visceral, trophic, reflex, etc., many combinations of symptoms result. These have been already described.

TABLE II.—PERIPHERAL NERVOUS DISEASES—TYPES AND SYMPTOMS.

Type.		Principal Symptoms.
Anæmia and Hyperæmia.		Sensory irritation: neuralgia, numbness.
Inflammation.	Multiple neuritis.	Acute onset, chronic course. At first sensory irritation, followed by loss; motor weakness; trophic and vasomotor disturbances; recovery one to two years.
	Mono-neuritis.	Acute or chronic: nerve irritation, later diminution and loss of function; trophic, vasomotor, and secretory disorders; recovery the rule, one-quarter to one and one-half years.

Degenerations.	Primary.	Gradual loss of function, little irritation, much trophic disturbance; very chronic; rarely a cure.
	Secondary.	Rapid loss of function, a little irritation; course chronic; cure frequent.
Destruction.		Sudden irritative symptoms, pain, spasm, etc.; then loss, like a secondary degeneration.
Tumors.	Single.	Irritative symptoms. Or no symptoms, chronic.
	Multiple.	
Functional neuroses.	Of irritative kind.	Neuralgia, paræsthesia, œdema, local asphyxia, spasm.
	Of depressive kind.	Occupation palsies, reflex palsies, etc.

CHAPTER X.

DISEASES OF THE SPINAL CORD.

Anatomy and Physiology.

Anatomy.—The spinal cord is a slender, cylindrically shaped organ. It is from 42 to 45.7 cm. (sixteen and one-half to eighteen inches) long, being shorter absolutely and relatively in women. Its weight is about thirty-three grams (one ounce).

It is suspended in the vertebral canal, where it reaches in all persons over one year of age as far down as the second lumbar vertebra. In new-born infants it extends to the third lumbar vertebra.

It is divided into cervical, dorsal, lumbar, and sacral portions, corresponding with the nerves it gives off. These are respectively four, ten and one-half, two, and one and one-half inches long (see Fig. 79). Its shape on cross-section is nearly round, except in the lower cervical region, where it is flattened antero-posteriorly. Its average diameter is 1 cm. (two-fifths of an inch). It has two swellings or enlargements, the cervical and lumbar. Their position, size, and extent are shown on the diagram. Its specific gravity is 1030.

It is surrounded by three membranes, all of which are continuous with the corresponding envelopes of the brain. They are the dura mater, the arachnoid, and the pia mater (Fig. 80).

The *dura mater* is the external covering. It is a long sac attached to the edge of the foramen magnum above and extending down until its walls fuse together at the level of the second sacral vertebra. Its cavity, therefore, is much longer than the spinal cord. It is attached to the bony canal at its lower end, and is held loosely by twenty-two lateral ligaments (*l. denticulata*) throughout its length.

The *arachnoid* is a thin, semi-transparent membrane lying loosely over the cord and roots.

Internally it is connected by numerous connective-tissue filaments with the innermost membrane, the *pia mater*. This latter is a thin vascular sheath applied closely to the cord and roots. The space between the dura and arachnoid is called the arachnoid cavity. It contains a very little cerebro-spinal fluid. That between the arachnoid and pia is called the subarachnoid cavity; it contains a good deal of cerebro-spinal fluid. Both cavities connect with those of the brain and probably with each other. The dura has a mechanical protective function, the arachnoid a serous and the pia a vascular function.

The spinal cord is movable in its canal to the extent of one-half to one inch.

The Nerve Roots.—The spinal nerve roots are covered with the pia and arachnoid. They pierce the dura in two places and unite to form a mixed nerve. The dura mater is prolonged over the nerves as they pass through it, forming a tubular sheath.

The anterior roots are the larger. At the point of exit of the nerves from the cord a slight depression is formed.

The Root Ganglia.—On each posterior root, *outside the dura*,

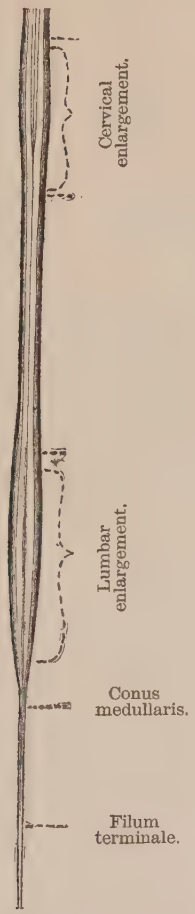


FIG. 79.—SHOWING THE RELATIVE SIZE OF THE DIFFERENT PARTS OF THE CORD (AFTER ALLEN).

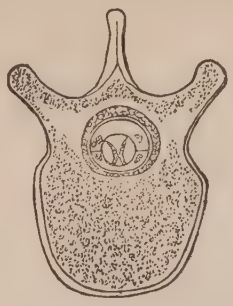


FIG. 80.—TRANSVERSE SECTION OF THE SPINE, showing the relations of the cord to the spinal canal (Keys and Retzius).

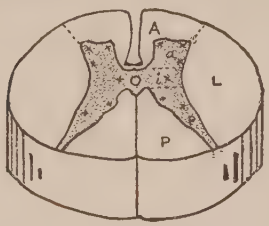


FIG. 81.—SHOWING THE RELATIONS OF THE GRAY MATTER TO THE WHITE. A, Anterior; L, lateral and P, posterior columns; the anterior (*a*) and posterior (*p*) horns and intermediate gray matter.

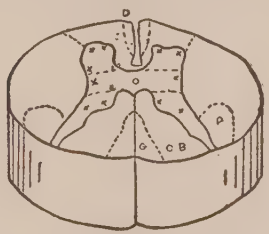


FIG. 82.—SHOWING FURTHER SUBDIVISIONS OF THE WHITE MATTER AND THE LOCATION OF THE DIFFERENT CELL GROUPS IN THE GRAY MATTER. D, P, Pyramidal tracts; CB, col. Burdach; G, col. Goll.

is a posterior spinal ganglion. The ganglia lie in the intervertebral canal, except those on the sacral nerves.

Fissures.—Throughout the whole length of the cord there are two median fissures, called the anterior and posterior.

Columns.—These fissures and the lines formed by the exit of the roots divide the cord into four columns—anterior, posterior, and two lateral.

The Composition of the Cord.—The cord is composed of white and gray matter. The white matter lies outside and is composed mainly of nerve fibres, the gray matter mainly of nerve cells. Each has also neuroglia, connective tissue, and blood-vessels. In the gray matter is a central canal lined with epithelial cells.

The gray matter is arranged, as shown in the figures, somewhat in the shape of a letter H. Its different parts are called the anterior and posterior horns and intermediate gray. At certain levels there are lateral horns.

The gray matter changes in shape at different levels of the cord. It is greatest in amount at the lumbo-sacral junction (23.33 sq. mm.); next at the cervical enlargement (sixth cervical) (17.32 sq. mm.). It increases in amount relatively to the white matter from above downward (Fig. 88).

The Commissure.—The gray matter of the two halves of the cord is connected by a bridge or commissure. The anterior part is composed chiefly of white medullated nerves, and is called the white commissure. The posterior is composed of very fine nerve fibres, mostly medullated collaterals, called the gray commissure. Between the two is the central canal, and surrounding it is the *central gelatinous substance*, composed of neuroglia.

The posterior horns reach to the periphery. They are divided, beginning from without, into the rim zone or Lissauer's column, the spongy zone, and the gelatinous substance of Rolando. The rim zone is composed of very fine nerve fibres; the spongy zone and gelatinous substance are composed chiefly of neuroglia * and small nerve cells.

To sum up, we have the gray matter—

Arranged in:

1. Horns: { anterior, lateral, posterior.
2. Intermediate gray.

Composed of:

a. A ground substance of neuroglia and connective tissue forming the substantia spongiosa.
b. Cell groups: internal, anterior, etc.
c. Plexuses of fine nerve fibres, *e.g.*, in the rim zone.
d. Masses of neuroglia:
  (1) The central gelatinous substance.
  (2) The gelatinous substance of Rolando and the spongy zone.
e. Blood-vessels and connective tissue.

Now, taking up some of these factors in detail, we find that

(*a*) The ground substance of the gray matter is made up of a fine meshwork of fibres which are the processes of neuroglia cells and of nerve cells. Besides this, there is some connective tissue, and there are prolongations from the base of the epithelial cells lining the central canal.

* Weigert's new stain seems to show that the substance is not neuroglia.

(*b*) The nerve cells are arranged in groups with the long axis parallel to that of the cord. The cells are surrounded by a rich plexus of cell-processes and nerves, as well as by the supporting neuroglia matrix, a little connective tissue, and many small blood-vessels. The cell groups are named in accordance mainly with their position—internal, antero-lateral, lateral, median, posterior or sensory spindle cell, and the cells of Clark's column (Fig. 83).

The cells are arranged in groups which overlap each other. Each group has the special duty of presiding over certain sets of muscles or other organs which have a common function.

The cells of the anterior horns are large in size, 35 to 100 μ ($\frac{1}{700}$ to $\frac{1}{250}$ in.); they are multipolar, having five or six processes, one of which is an axis-cylinder process. The cells in the central parts of the horn are the smaller; the cells in the lumbar swelling are largest, because they are connected with long nerves. The cells of the cervical swelling are next in size. The cells of the posterior horn are small and multipolar.

The cells of Clark's column are bipolar, 30 to 60 μ ($\frac{1}{850}$ to $\frac{1}{400}$ in.) in diameter, and are arranged with their long diameter parallel to the axis of the cord. They are grouped together in a kind of nest at the inner and central part of the posterior horn (see Fig. 82). Clark's column is most distinct from the eighth dorsal to the second lumbar nerves, but extends up as far as the last cervical. An analogous group of cells is found at the level of the second and third sacral nerves.

The spindle-cell group consists of small cells lying in the intermediate gray matter at the base of the posterior horns. There are other minor groups of cells which it is not necessary to describe here.

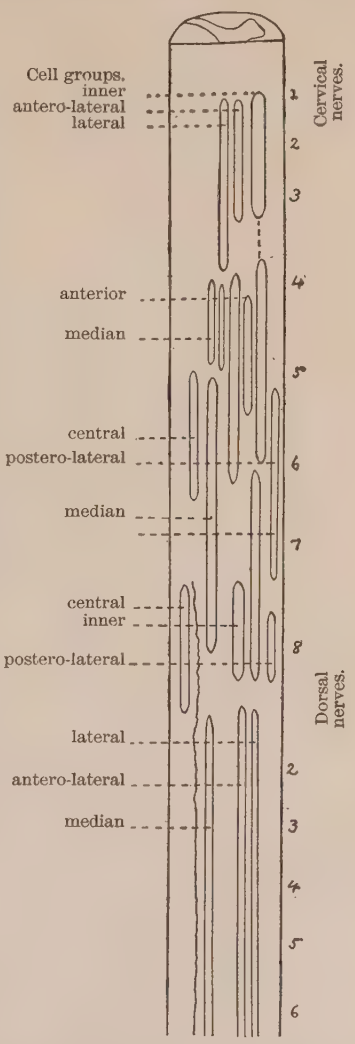


FIG. 83.—SHOWING THE ARRANGEMENT OF CELL GROUPS OF ANTERIOR HORNS AND INTERMEDIATE GRAY IN THE UPPER HALF OF THE CORD. Showing also the levels to which each group extends.

The white matter of the cord is composed mainly of nerve fibres, running in a supporting net-work of neuroglia, connective tissue, and blood-vessels. Surrounding it, and lying just beneath the pia mater, is a thin layer of neuroglia 5 to 50 μ ($\frac{1}{5000}$ to $\frac{1}{500}$ in.) thick. The nerve fibres are medullated, but have no neurilemma, and but few, if any, nodes of Ranvier. There are two kinds. the large (81 to 20 μ) and the small (2 to 3 μ in diameter). The small fibres make up the postero-internal (Goll's) column entirely, and are numerous in the deep part of the lateral columns, but they are found in all regions. The fibres run up and down for the most part. They are arranged in columns, the division being based

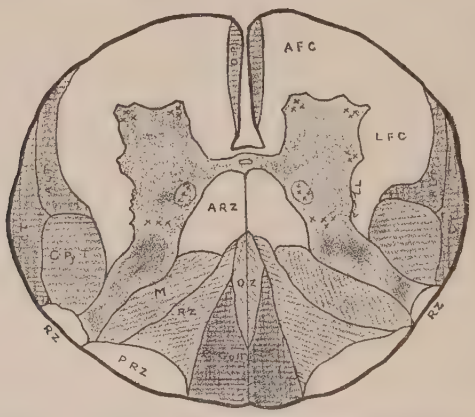


FIG. 84.—SECTION OF SPINAL CORD, showing complete subdivision of white columns into:

Anterior columns: DPy, Direct pyramidal tract. AFC, Anterior fundamental column.

Lateral columns: LFC, Lateral fundamental column. LL, Lateral limiting layer. CPyT, Crossed pyramidal tract. CT, Direct cerebellar tract. ALT, Antero-lateral ascending tract.

Posterior columns: Column of Goll. Column of Burdach. RZ, Rim zone, or Lissauer's column.

ARZ, Anterior root zone. MRZ, Middle root zone. OZ, Oval zone. PRZ, Posterior root zone.

partly on anatomical, partly on physiological, and partly on embryological grounds.

Anatomically there is a simple and natural division, which we have already given, into the anterior, lateral, and posterior columns, the divisions being made by the median fissures and the roots of the nerves (see Fig. 81).

On physiological and embryological grounds the columns are further subsided as follows:

The anterior columns are divided into: Direct pyramidal tract. Anterior fundamental column.

The lateral columns are divided into:
- Lateral fundamental columns.
- Lateral limiting layers.
- Crossed pyramidal tracts.
- Direct cerebellar tracts.
- Antero-lateral ascending tracts, or Gowers' column.

The posterior columns are divided into:
- Postero-internal column, or column of Goll.
- Postero-external columns, or column of Burdach, divided into:
  - Anterior root zone.
  - Middle root zone.
  - Oval root zone.
  - Posterior root zone.
- Rim zone, or column of Lissauer.

The fibres which make up these columns are of two kinds—*long* or projective, *short* or associative.

The *long* fibres connect the different levels of the cord with the brain, and the different spinal nerve roots with nuclei in the upper part of the cord.

The *short* or associative fibres connect different levels of the cord with each other, and also connect the two halves of the cord at the same levels.

The names of the long-fibre tracts are the direct and crossed pyramidal, the direct cerebellar, the antero-lateral ascending, and the postero-internal or column of Goll.

The *direct pyramidal tract* lies along the anterior median fissure and extends down as far as the lower part of the dorsal cord. Its fibres cross over in the anterior commissure at various levels and connect with the cells of the anterior horns.

The *crossed pyramidal tract* extends down the whole length of the cord and sends its fibres to the anterior horns of the same side.

Both of the above tracts are continuations of the anterior pyramids or motor tracts of the medulla. These pyramids divide at the lower end of the medulla, about ninety per cent of fibres crossing over to form the crossed pyramidal tract and ten per cent continuing on the same side. Some of the fibres of the crossed pyramid redecussate (in lower animals) and enter the pyramidal tract of the side on which they started (Fig. 88, PT).

The *direct cerebellar tract* begins at the level of the first lumbar nerves. Its fibres originate in the vesicular column of Clark. They pass up to the cerebellum and go chiefly to the vermis. Most of them then cross over and enter the red nucleus (Fig. 88).

The *antero-lateral ascending column* extends nearly the whole length of the cord. Its fibres come from the posterior commissure and the sensory cells of the opposite posterior cornu. They pass up and end in the lateral nucleus (Fig. 88, ALC).

The *postero-internal column*, or column of Goll, is composed of fibres which originate in the ganglia of the posterior roots, pass inward, and without crossing ascend, to end in a nucleus at the upper limit of the cord, the nucleus of Goll's column (postero-internal nucleus). The column extends the whole length of the cord. It is very small in the sacral region, but increases in size as it passes up (Fig. 88, G).

There are a few long fibres scattered in the anterior and lateral ground bundles. They degenerate down and are called the *antero-lateral descending tract.*

The names of the short-fibre columns are the anterior and the lateral fundamental columns, the lateral limiting layer, and the column of Burdach. This latter column contains in its cervical part some long fibres which end in a nucleus at the upper limit of the cord, the postero-external nucleus, or nucleus of the column of Burdach (Fig. 82, CB).

The Relations of the Different Parts of the Spinal Cord to the Peripheral Nerves, to the Brain, and with Each Other.—I will begin with a description of the way in which the anterior and posterior nerve roots are connected to

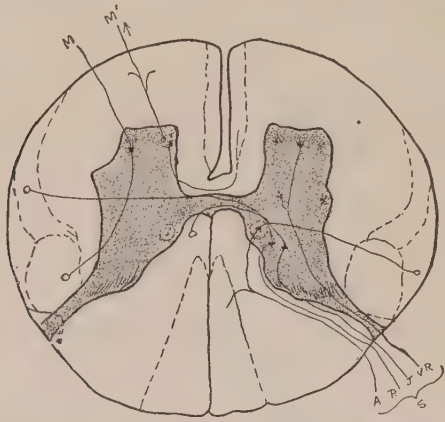


Fig. 85.—Showing the Relations of the Anterior Roots M, M′ to the Anterior Horns; of the Anterior Horns to the Crossed Pyramidal Tracts and Posterior Roots. Showing also the relations of the posterior root fibres: T, to column of Goll and Clark's column; P, to sensory cells and posterior column; J, to sensory cells and antero-lateral ascending column; R, to anterior horns. S, Sensory root; A, muscle sense; P, pain and temperature sense; T, tactile sense; VR, visceral and reflex fibres.

the cord; then describe the mode in which the different columns and cell groups are connected with each other; and finally I will indicate briefly the connections of the cord with the brain.

The *anterior nerve roots* are connected directly with the anterior horn cells, of which they are processes.

The cells of the anterior horn are surrounded by two sets of "end-brushes," one coming from the pyramidal tracts, the other from the posterior horns and roots. Thus these cells are in relation with impulses from the brain and from the periphery.

The *posterior nerve roots* originate chiefly in the spinal ganglia. On entering the cord the fibres divide like a T and pass up and down for one or two inches. They then enter (1) the column of Goll, (2) the anterior or posterior commissure, or (3) the posterior horn. They all send off collaterals, and terminate eventually in end-brushes surrounding nerve cells.

There are at least three groups of nerve fibres which enter by

the posterior roots and make different connections with the cell groups or columns:

1. An innermost set. These pass across the postero-external column and enter the median or Goll's column, which they ascend, to end in the nucleus.

2. A median set. These pass along the inner side of the posterior horn, and end either (*a*) in cells of the deeper part, (*b*) in the spindle-shaped cells, or (*c*) go to the anterior horn; still others (*d*) cross over in the commissure to enter the antero-lateral tract.

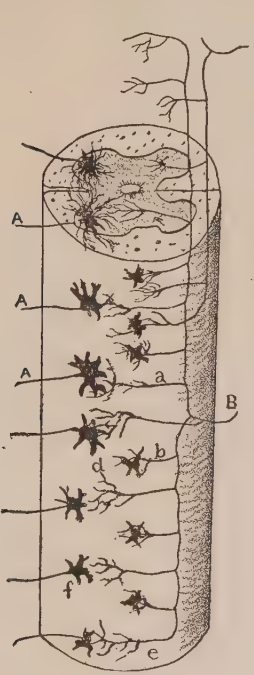


Fig. 86.

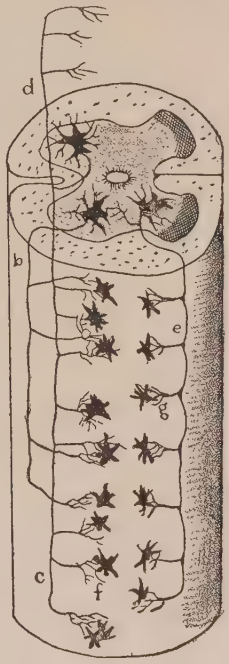


Fig. 87.

Fig. 86.—Showing the Connections of the Anterior and Posterior Roots and Cornua with Each Other (Cajal). A, Anterior root; B, posterior root; *a*, collaterals; *d*, end-brushes.

Fig. 87. — Showing the Association or Short-Fibre System of the Cord (Cajal). *a*, Anterior cornua cell; *b*, *c*, *d*, association fibres; *e*, posterior association fibres.

3. An outer set. These are very fine fibres which enter the tip or outer part of the posterior horn, and then run up and down, forming the rim zone. They eventually connect in the usual way with the sensory cells of the posterior horn (see Fig. 85, A, R).

The different parts of the spinal cord are connected by the short fibres which unite different levels of the cord, and by commissural fibres which unite the different halves of the cord. These

short and commissural fibres originate in groups of nerve cells lying in the central parts of the gray matter and called associative nerve cells. They are small and multipolar. Some are distributed sparsely in the white columns. Fibres arise from them, run in the commissures and short-fibre tracts, and end in brushes which put the fibres in relation with various cell groups (Figs. 86, 87).

Physiology.—The detailed facts regarding the functions of the spinal cord may be gotten in physiological text-books. I shall give only those bearing more or less directly on the localization of the functions. The spinal cord is a conductor and centre of nervous action. It represents the lowest evolutionary level of the development of the nervous system. Its functions, so far as they are independent, are stable and well organized, but of a mechanical and relatively simple order.

Functions of the White Columns.—The white matter is a conductor of nerve impulses, and its functions are relatively simple. We have only to study the direction and kind of impulses carried by its various columns. The *direct* and *crossed pyramidal tracts* carry motor impulses downward from the brain. A few fibres (two to ten per cent) connect with muscles of the same side; so that in hemiplegia there is usually some weakness on the healthy side. These tracts normally exercise a continual inhibitory influence on the motor cells of the anterior horns; so that when destroyed there develop spasmodic conditions of the paralyzed part.

The anterior ground bundle, *lateral ground bundle*, and the *lateral limiting layer* have the function of associating different levels of the cord and of connecting it also with nuclei in the medulla and centres in the cerebellum.

The *columns of Goll* conduct special sensations from the muscles, articulations, and tendonous sheaths via the root on the same side. When diseased, there is a loss of the sense of position of the limbs, of the power of estimating weights, and of co-ordination of muscular effort (ataxia). The fibres cross over in the medulla.

The *columns of Burdach* conduct to a certain extent tactile sensations coming in from the opposite posterior root. They also contain many associative fibres, connecting different levels. Pain-sense fibres and excito-reflex fibres from the posterior roots run through these columns to reach the commissure or the anterior horns; other fibres run through it to Clark's column and to the column of Goll. Hence it is a pathway for all kinds of afferent impulses. When diseased, there may be pain, anæsthesia, ataxia, and loss of reflexes. The fibres cross over at once to the opposite side.

The *antero-lateral ascending tract* conducts sensations of pain and temperature, coming in from the opposite side, through the anterior and posterior commissure.

There are considerable variations in the paths of conduction of tactile temperature and pain sensations, and their exact position is not positively known. In transverse lesions of the cord these tracts do not generate upward so completely or uniformly as do secondary degenerations of other long-fibre tracts. Hence they probably receive some interruptions in their course.

Automatic Centres.—The nerves and cells of the cord are arranged in complex groups which preside over certain functions or respond in a definite way to certain stimuli. These are

called the spinal automatic centres. They are the cilio-spinal, secretory, vasomotor, genital, vesical, and rectal. The important parts of these centres lie deep in the gray matter on either side of the central canal, but nearer the base of the posterior horns. Lesions of the white matter, or of the anterior or posterior horns, do not directly affect them.

The *cilio-spinal centre* reaches from the seventh cervical to the second dorsal segment, inclusive. Its stimulation causes the pupils to contract.

The *genital centres*, including those for erection and ejaculation, reach from the first to the third sacral segment, inclusive.

The *bladder* and *rectal centres* are in the fourth and fifth sacral segments, extending up and down a short distance, the bladder being perhaps a little lower.

The spinal *vasomotor centres* extend from the second dorsal to the second lumbar segments.

The vaso-dilator nerves pass out by the anterior, the constrictor by the posterior, root (Gaskill).

The *gray matter* contains chiefly cell groups which act as centres and distributors of nerve impulses. In the *anterior horns* the cells have a motor and trophic function. The larger cells are at the outer parts of the horn and send fibres to the larger skeletal muscles. The more central cells are connected with small muscles and those having more delicate functions and adjustments. In the still more central and intermediate parts, also, are separate trophic cells for the muscles, bones and joints, and cell groups which preside over vasomotor and secretory functions. Among these groups are the *spindle-shaped cells*, which send fibres to the vaso-constrictors (Gaskill), through the anterior (Hill) and perhaps posterior roots (Gaskill). The cells of Clark's column receive fibres from the viscera and blood-vessels. Impulses pass to these cells and thence to the direct cerebellar tract and cerebellum. Their function is to conduct impulses from the viscera relating to equilibrium and sense of posi-

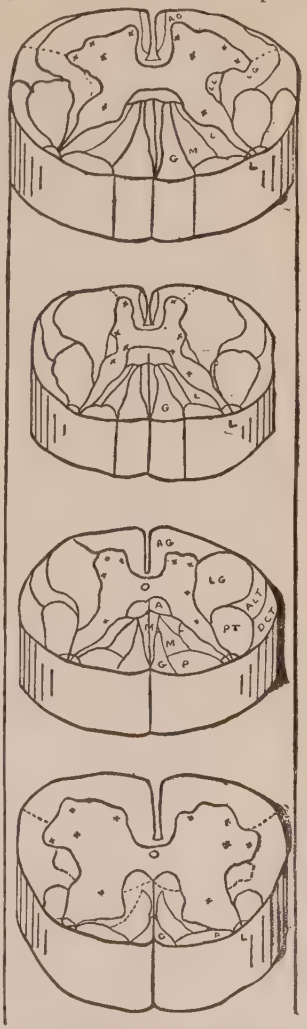


FIG. 88.—SHOWING THE RELATION OF THE ARRANGEMENT OF THE GRAY MATTER, CELL GROUPS, AND WHITE COLUMNS AT FOUR DIFFERENT LEVELS. Namely, mid-cervical, mid-dorsal, lower dorsal, and upper sacral. (Drawn from specimens.)

tion. They are analogous to the fibres of the column of Goll. According to Gaskill, Clark's column is a centre for the vaso-dilators. This is unlikely.

The *cells of the posterior horns* are sensory in function and are connected with the tactile, pain, temperature, and reflex fibres of the posterior roots.

The *trophic centres for the joints, bones, and skin* apparently lie in the posterior horns. Their fibres pass out by the posterior roots.

Topography and Localization.—The neurologist and surgeon need to know, for purposes of diagnosis:

1. The relation of the special nerve roots, at their point of origin, to the spinous processes. This is shown in the figure (p. 156). In general it will be seen that the different pairs of nerve roots arise opposite the spinous process of a vertebra one or two segments above those between which it makes its exit. Thus the sixth cervical originates opposite the fourth cervical spine, the sixth dorsal between the third and fourth dorsal spines, the first lumbar between the eleventh and twelfth dorsal spines. There is considerable variation in these relations, as the diagram from Reid shows (Fig. 72).

2. The next points desired are the special function of each pair of nerve roots anterior and posterior, and the level of the various centres in the cord. This is shown in the following table, based on that originally devised by Starr, modified by Mills and myself from personal experiments and the clinical and pathological observations of Thorburn and others.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflex and Centres.	Sensation.
First cervical.	Rectus laterales. Rectus capitis. Anticus and posticus. Sterno-hyoid. Sterno-thyroid.		
Second and third cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	*Hypochondrium* (?). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
Fourth cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	*Pupillary* (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external musculo-cutaneous, cutaneous.)
Fifth cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder-blade. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	*Scapular* (fifth cervical to first dorsal). Irritation of skin over the scapula produces contraction of scapular muscles. *Supinator longus.* Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)

Segment.	Muscles.	Reflex and Centres.	Sensation.
Sixth cervical.	Deltoid. Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators. Rhomboid. Latissimus dorsi.	*Triceps* (fifth to sixth cervical). Tapping elbow tendon produces extension of forearm. *Posterior wrist* (sixth to eighth cervical). Tapping tendons causes extension of hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly external cutaneous, internal cutaneous, radial.)
Seventh cervical.	Triceps (long head). Extensors of wrist and fingers Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	*Anterior wrist* (seventh to eighth cervical). Tapping anterior tendons causes flexion of wrist. *Palmar* (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index, and one-half middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)
Eighth cervical.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand muscles.	.....	Ulnar area of hand, back, and palm, inner border of forearm. (Internal cutaneous, ulnar.)
First dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar muscles.	.....	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)
Second dorsal.	.....	.....	Inner side of arm near and in axilla. (Intercosto-humeral.)
Second to twelfth dorsal.	Muscles of back and abdomen. Erectores spinæ.	*Epigastric* (fourth to seventh dorsal). Tickling mammary region causes retraction of the epigastrium. *Abdominal* (seventh to eleventh dorsal). Stroking side of abdomen causes retraction of belly. Vasomotor centres. Second dorsal to second lumbar.	Skin of chest and abdomen, in bands running around and downward, corresponding to spinal nerves. Upper gluteal region. (Intercostals and dorsal posterior nerves.)
First lumbar.	None.	*Cremasteric* (first to third lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Ilio-hypogastric, ilio-inguinal.)
Second lumbar.	Vastus internus.	*Patellar*. Striking patellar tendon causes extension of leg.	Outer side and upper front of thigh. Lumbar region. (Genito-crural, external cutaneous.)
Third lumbar.	Sartorius; adductors of thigh. Flexors of thigh.	.....	Front and outer side of thigh. Inner side of leg and foot.
Fourth lumbar.	Extensors of knee. Abductors of thigh.	*Gluteal* (fourth to fifth lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg, and foot. (Internal cutaneous, long saphenous, obturator.)

Segment.	Muscles.	Reflex and Centres.	Sensation.
Fifth lumbar.	Outward rotators. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	*Achilles tendon.* Over-extension causes rapid flexion of ankle, called ankle clonus.	Back of thigh and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculo-cutaneous, plantar.)
First and second sacral.	Calf muscles. Glutei. Peronei. Extensors of ankle. Small muscles of foot.	*Plantar* (fifth lumbar to second sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back of buttock and thigh, side of leg and ankle; sole; dorsum of foot.
Third, fourth, and fifth sacral.	Perineal. Muscles of bladder, rectum, and external genitals.	Genital centre. Vesical centre. Anal centre.	Circumanal region, anus, rectum, penis, urethra, vagina, perineum. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)

THE BLOOD SUPPLY OF THE SPINAL CORD is a subject of great practical importance; and as our knowledge of it has lately been increased, I shall present the matter here in some detail.

The spinal cord is supplied with blood by branches from the vertebrals, ascending cervical, and superior intercostal arteries above, and by the dorsal intercostal, lumbar, and sacral arteries below. These send off small branches which enter the spinal canal through the foramen magnum above and the intervertebral foramina at the sides; they pierce the dura mater and are distributed on the pia mater and in the cord. The arteries that thus supply the cord are these:

Primary Arteries.	Origin from	Ending in
Anterior spinal.....	Vertebral (from subclav.).	Anterior median spinal artery.
Posterior spinal.....	Vertebral.	
Lateral spinal.....	Vertebral.	
Lateral spinal..... ..	Ascending cervical (from subclav.).	Anterior and posterior spinal root arteries.
Lateral spinal.	Superior intercostal (from subclav.).	Anterior and posterior spinal root arteries.
Lateral spinal..... ...	Thoracic intercostal (from aorta).	Anterior and posterior spinal root arteries.
Lateral spinal	Lumbar (aorta). Lateral sacral (from int. iliac).	Anterior and posterior spinal root arteries.

The anterior spinal arteries are branches of the vertebrals. They unite to form the anterior median artery, which runs down the whole length of the cord, receiving re-enforcements from the lateral arteries (Fig. 89). The *anterior spinal* arteries themselves nourish only a few upper segments of the cord. The anterior median artery is not, as has been taught, a true prolongation of the anterior spinals, but is really made up by the lateral spinals. In other words, the vertebral artery through its branches only nourishes the upper cervical region of the cord. The *posterior*

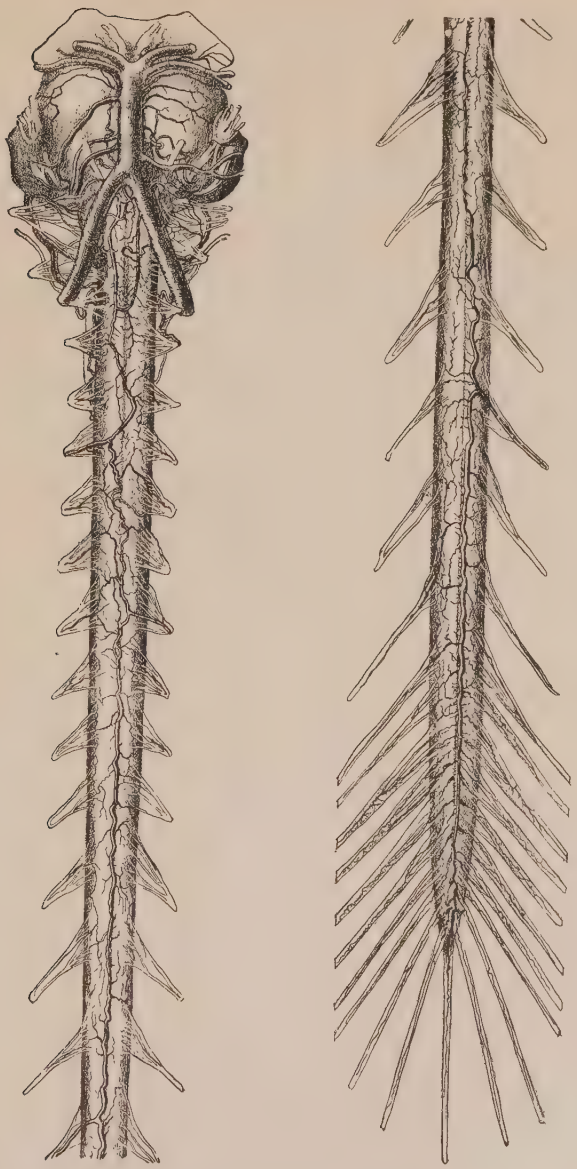


FIG. 89.—THE SPINAL CORD, ANTERIOR SURFACE, showing the nerve roots, root arteries, and anterior plexus (Kadyi).

spinal arteries are smaller than the anterior and unite on the posterior surface of the cord. They do not continue down as a posterior median artery—there is no such artery; but they help to form two plexuses on the postero-lateral surfaces of the cord.

The *lateral spinal* arteries are derived from branches of the subclavian artery as far down as the second dorsal root; below this point by the thoracic and abdominal aorta and the internal iliac. It is an interesting fact that at or a little below the point where the blood supply changes from the subclavian above the heart to the aorta below, pathological disturbances frequently occur (transverse myelitis).

Root Arteries.—The lateral spinal arteries, after they enter the spinal canal, are called the root arteries. They pierce the dura mater and pass, some along the posterior and some along the anterior roots, to the cord. There are about eight anterior root arteries (five to ten) and about sixteen posterior root arteries (see Figs. 89, 90). The anterior arteries are twice as large (one millimetre in diameter) and one-half as numerous as the posterior. The root arteries of the cervical region are rather the more numerous. There is a large and constant anterior root artery in the

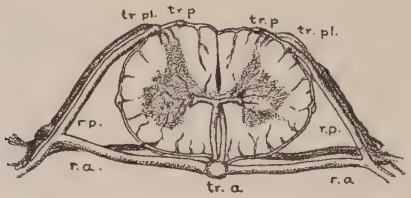


Fig. 90.—Arterial Supply of Cord. *r.a.*, Anterior root artery; *r.p.*, posterior root artery; *tr.a.*, anterior plexus; *tr.p.* and *tr.pl.*, posterior and lateral plexuses (Kadyi).

dorso-lumbar region. The last two lumbar, the five sacral nerves, and the unpaired coccygeal nerve when it exists are accompanied by small root arteries which do not reach up to the cord itself. The lower part of the spinal cord is supplied by large root arteries from the lateral spinal arteries. Hence the theory of Moxon that the circulation here is feeble is not supported by Kadyi's investigations.

The Plexuses.—The anterior root arteries pass to the anterior median fissure, and then divide, partly to form the anterior median artery and partly to form a rich plexus between the anterior roots: this is called the *anterior arterial plexus.* The posterior root arteries subdivide before they reach the cord, and send twigs to its lateral and posterior surfaces which form the *postero-lateral arterial plexus.* The posterior root arteries do not anastomose to any extent with each other or form a posterior spinal artery, as is done by the anterior root arteries. There are therefore three relatively independent arterial plexuses: The anterior plexus; the two postero-lateral plexuses.

Veins.—The veins of the spinal canal outside the dura mater have valves, those within it have none. The veins reach the pia mater and cord by passing along the nerve roots. Hence we have anterior and posterior *root veins*, corresponding to the root

arteries, but more numerous, there being a total of forty or fifty. The anterior root veins are more numerous than the posterior, but smaller (twenty-five to twenty). The veins are a little larger than the arteries, the anterior veins being one-half to one millimetre, the posterior one and one-half to two millimetres, in diameter.

Thus we see that the posterior surface of the cord has more and smaller arteries, fewer but larger veins. The posterior surface is on the whole more richly supplied with veins, the anterior surface with arteries. The lateral surfaces are the least vascular.

	Number.	Size.
Anterior root arteries	5 to 10.	1 mm.
Anterior root veins	25 to 30.	½ to 1 mm.
Posterior root arteries	16.	½ mm.
Posterior root veins	20 to 25.	1½ to 2 mm.

Vessels of the Cord Substance.—The cord is supplid by (1) central arteries which are branches of the anterior median, and by (2) peripheral arteries which come from the plexuses on the pia mater. These two systems have been called also the centrifugal and centripedal respectively. They are not absolutely independent, but are in a good measure so. The central arteries nourish chiefly the gray matter, the peripheral arteries the white. Both systems are made of "end arteries," *i.e.*, they do not anastomose with each other. Neither the central nor peripheral arteries are distributed in accordance with anatomical relations or physiological functions. Each cell group, for example, has a vascular supply from several sources.

The *central arteries* are given off from the branches of the anterior median at the bottom of the median fissure and number about two hundred, each spinal segment having six or seven. The accompanying central veins are small and their total capacity is less than that of the arteries, so that the central arterial pressure must be high, on account of the poor venous outlet (Kadyi). Some of the blood escapes by the peripheral veins.

The *peripheral* arteries pass into the spinal cord for the most part along the various connective-tissue septa. There they branch and supply chiefly the white matter. They supply the apex and some of the deeper substance of the posterior horns and Clark's columns. The arteries of the posterior septum are the largest and most numerous, often reaching to the gray commissure. The peripheral arteries are smaller than the corresponding veins (.04 to .2 mm.). The relation is just the reverse, therefore, of that of the central arteries and veins. The peripheral arteries are small, and after passing into the cord branch into minute vessels which pass up and down and soon become capillaries. The central arteries, on the other hand, continue large, and run up and down some distance before they are subdivided into capillaries.

To sum up: the arteries predominate in total capacity in the anterior plexus and central arteries. The veins in the posterior plexuses and peripheral veins. The central arteries are larger and longer than the peripheral. Hence the blood circulates more quickly and under greater pressure in the central gray of the cord. Conditions of enfeebled circulation would affect the posterior columns and roots more than the anterior and central parts of the cord.

CHAPTER XI.

THE DISEASES OF THE SPINAL CORD.

THERE are about thirty diseases which may be classified as belonging to the spinal cord. Most of these are organic in character and come under the head of inflammatory and degenerative or system diseases. Functional disorders referable to the cord alone are rare; while of organic diseases, those that result from injury and inflammation are the most common.

Etiology.—The causes of spinal-cord diseases can nearly all be formulated under the heads of injury, exposure, poisons, autotoxæmiæ, infections, and excessive functioning. Persons of middle life are the most predisposed, while heredity does not play an important part.

Symptoms.—The symptoms of all disorders of the nervous centres can be included under the heads of those of irritation, depression, and perversion. The principal irritative symptoms in spinal-cord disease are pains and paræsthesiæ of the back and limbs, hyperæsthesia and feelings of constriction around the waist, rigidity, spasms, exaggerated reflexes, and irritability of the visceral and vascular functions. The principal symptoms of depression and destruction are anæsthesia, ataxia, paralysis, wasting, and loss of power over visceral centres. The common form of paralysis in spinal-cord disease is paraplegia, in brain disease hemiplegia, in multiple neuritis quadruplegia. Symptoms of irritation and depression often accompany each other. The more superficial and meningeal the disease, the more are the symptoms irritative; the more central and myelonic the trouble, the less the irritation and the more the paralysis and visceral disturbance. Thus meningitis, meningeal tumors, and hemorrhages are extremely painful; while central myelitis is almost painless.

Pathology.—Inflammations of the meninges of the cord are not rare; the opposite is true of primary inflammations of the cord itself. As will be shown later, most of the diseases that used to be called chronic myelitis are secondary to injuries and softenings. Degenerative diseases of the cord, which include such affections as locomotor ataxia and progressive muscular atrophy, used to be called "system diseases" because they affected certain long-fibre tracts or systems of cell groups. The name implies re-

strictions which are not justified in fact, and it can only be retained as a matter of convenience. Secondary degenerations alone are aways systemic. The cord is relatively free from abscesses hemorrhages, and tumors.

Diagnosis.—In making a diagnosis of spinal-cord diseases, one is most helped by a thorough knowledge of the cord functions. In no part of the economy do physiology and anatomy point out more clearly the path to the clinician.

Prognosis.—The spinal-cord tissue once destroyed can never be renewed, or only to a limited extent, and that as regards the nerve fibres, not the cells. It has considerable power of adjusting itself to damage; but, on the other hand, serious injury is likely to extend by the process of secondary degeneration. Functional diseases, vascular and nutritive disturbances, of the cord can never be so severe or chronic as to exclude the possibility of recovery.

The special diseases of the spinal cord are the following:

1. *Malformations:* Myelocele, meningo-myelocele (spina bifida), meningocele, heteropia, amyelia, micromyelia, macromyelia, double cord.
2. *Vascular Disorders:* Anæmia, hyperæmia, hemorrhage, endarteritis with aneurism, embolism or thrombosis, œdema. *Secondary* to these conditions are *softenings* and *sclerosis.*
3. *Inflammations:* Meningitis, myelitis, abscess. Secondarily, softenings, sclerosis.
4. *Degenerations:* Primary: locomotor ataxia, combined scleroses, hereditary scleroses, progressive muscular atrophy, and allied types.
5. *Tuberculosis:* Miliary and solitary.
6. *Syphilis:* Gumma, meningo-myelitis, vascular disease.
7. *Tumors.*
8. *Functional and toxic disorders.*

MALFORMATIONS.

Spina Bifida (Rachischisis Posterior).

Spina bifida is a congenital hernia of the spinal membranes, and sometimes of the cord, through a cleft in the vertebra caused by absence of the vertebral arches. It is really a malformation of the vertebral canal rather than of the cord.

Etiology.—The condition is not very rare, about 1 child in 1,200 (French statistics) being affected. It is often associated with hydrocephalus or with some other defect in development, such as ventral hernia, imperforate anus or pharynx. Hereditary influence is sometimes a factor. It is a true developmental defect,

and is not due to a primary dropsy of the cord, as was once taught.

Forms.—There are three varieties described:

1. Spinal meningocele is a condition in which the spinal membranes alone protrude into the sac.

2. Spinal meningo-myelocele is a form in which the membranes and cord both protrude.

3. Syringo-myelocele (hydrorachis interna) is a form in which the fluid is in the central spinal canal, and the inner lining of the sac is formed by the meninges and thinned-out spinal cord.

Anatomy.—The first two forms are the most common and are called hydrorachis externa. The fluid here lies in the subarachnoid sac, and hence the wall of the protruding cyst is lined with

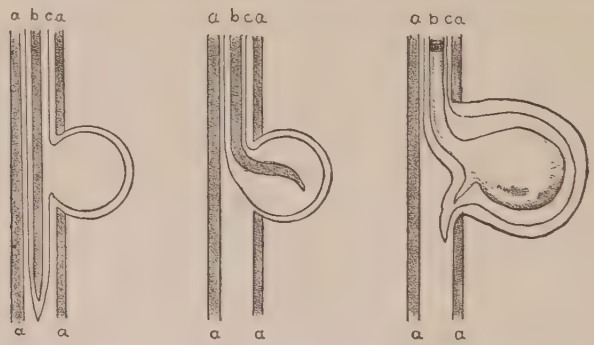


FIG. 91.—MENINGOCELE. MENINGO-MYELOCELE. SYRINGO-MYELOCELE.
a, Vertebral walls; *b*, cord; *c*, membranes.

the dura and arachnoid. The nerves and cord protrude into the sac in two-thirds of the cases (forming a meningo-myelocele), but in some of these only a few nerves are found. These structures, when present in the sac, lie on its *posterior and median surface*, sometimes causing a depression there. The tumor contains cerebro-spinal fluid, and occasionally connective tissue and fat (Fig. 91).

Symptoms.—Spinal bifida occurs almost always in the lumbar and sacral region, the reason being that the lamina here are the last to solidify. Usually but two or three vertebræ are involved. The tumor varies in size from 3 cm. (one inch) to 15 cm. (six inches) in diameter and may have a broad base or be pedunculated. The outer skin is often glossy, or tough, thickened, or ulcerated.

Children with spina bifida are usually feeble, badly nourished, and poorly developed mentally. Paraplegia occurs in half

the cases, sometimes with anæsthesia and involvement of the sphincters. Talipes occurs quite often.

The *prognosis* is grave. Most cases die unless treatment is applied, and even then the prospect is not very good.

The *diagnosis* is easy. It is generally only necessary to exclude congenital tumors which happen to be located in the lumbo-sacral region. The most important question to decide is whether the cord and nerves are present in the sac. This may be assumed as probable if there is much paraplegia, anæsthesia, and sphincter trouble. The introduction of an insulated needle connected with an electric battery may be tried.

FIG. 92.—SPINA BIFIDA.

The *treatment* is strictly surgical. At present, injections of Morton's fluid (iodine, gr. x.; potas. iodid., gr. xxx.; glycerin, ℥ i. Dose, ʒ i.) have been most successful. These injections should be made in the lateral portion of the sac, and the child should be kept on the back. Puncture and withdrawal of fluid with compression is not a justifiable operation The ligature or opening and excision of the sac are dangerous, especially if, as is often the case, part of the cord and nerves lie in the sac. In recent years, surgical results have been more favorable and warrant serious consideration. No surgical treatment should be attempted, however, until two or three months after birth.

HETEROPIA is a rare malformation in which masses of gray matter are found in abnormal situations. A false heteropia may be caused (Van Giessen) by manipulation of the cord in its removal after death. The displaced masses consist of nerve cells or neuroglia.

AMYELIA or absence of the spinal cord can only exist when the brain is absent; but absence of the brain may occur without absence of the cord. In amyelia the spinal nerves are usually present. Amyelic monsters cannot live.

DOUBLE CORD is a very rare defect and involves only part of the cord, except in cases in which there is a double vertebral canal.

DOUBLE CENTRAL CANAL is not rare. It usually involves only a part of the cord. The two canals are side by side.

ASYMMETRY of the cord, usually due to abnormality in the course of the pyramidal tracts, is not extremely rare.

SPLITTING OF THE CORD and defects in development at special levels are occasionally observed.

MICROMYELY is a condition in which the spinal cord is abnor-

mally short or small in size, and is not a very rare anomaly. The normal adult cord has a diameter in its various parts of 6 to 9 mm. (dorsal), 8 to 11 mm. (upper cervical), 15 mm. (cervical swelling), and 12 mm. (lumbar).

Spinal Hemorrhage (Spinal Apoplexy).

This general name may be given to (1) spinal meningeal hemorrhage or hæmatorhachis, and (2) hemorrhage into the cord substance, or hematomyelia.

1. Spinal meningeal hemorrhage is far the most common form. It may be outside or inside of the dura, the former being rather oftener seen.

Etiology.—It occurs in newly born children and in adults, and is more common in men than in women. Injuries, falls, fractures of the spine are the most frequent exciting causes. Severe convulsions from epilepsy, eclampsia, tetanus, chorea, or strychnia may be a cause, also severe muscular exertion. Purpura and the blood states following malignant infectious fevers, bursting of an aortic or vertebral aneurism and cerebro-spinal meningitis are rare causes.

Symptoms.—In small hemorrhages there may be no symptoms. In large effusions there are sudden, very severe pains in the back, extending into the limbs, numbness, tingling, hyperæsthesia, and muscular spasm, especially of the back muscles. Later there may be weakness or paralysis and anæsthesia, with disorder of the visceral centres. The symptoms reach their height usually in a few hours. Then amelioration may occur, followed by slow recovery or with symptoms of chronic meningitis. Rarely death occurs early from exhaustion.

Diagnosis.—A history of injury, child-birth, sudden onset of attack, with symptoms of pain and irritation which rather rapidly subside, point to extra-dural hemorrhage. In hæmatomyelia there is less pain and irritation, but more profound paralysis and anæsthesia. The same is true of crush of the cord from fracture or dislocation. In tetanus there is a slower development of the symptoms, and trismus is present.

The *prognosis* is grave in severe cases; but if the patient survives three or four days, the prospect of partial or nearly complete recovery is good.

The *treatment* is perfect rest in bed and the administration of remedies to move the bowels and relieve pain; leeches and other local applications are of doubtful value. It is of no use to give styptics except in purpura, when mineral acids or chloride of calcium may be tried. Later, one may give iodide and mercury and use blisters to the back.

Hemorrhage into the Substance of the Cord (Hæmatomyelia).—*Etiology.*—The condition is not very rare. It may be primary from disease of the blood-vessels or purpura hæmorrhagica; or it may be secondary to myelitis and tumors. Primary hemorrhage occurs sometimes in infancy, but usually in males between the twentieth and fortieth year. Injuries, overexertion, exposure, excessive coitus (Gowers), and convulsions are causes.

The *symptoms* develop rapidly, with at first feelings of numbness or weakness for one or two hours or longer. Then there is a sudden paraplegia, with anæsthesia or ataxia or both. The sphincters may be paralyzed; the urine has to be drawn. The reflexes may be abolished at first, but soon return and become exaggerated. There is considerable pain in the back. If the lesion is high up, the arms and thorax are involved. The acute symptoms begin usually to subside at the end of seven to ten days and the disease takes the character of a chronic myelitis. If improvement does not occur, evidences of acute myelitis appear and the patient dies.

Pathology.—The vessels involved are the central arteries, which supply the gray matter and are under relatively high pressure. The rupture of the vessel, when due to disease, is caused by a fatty degeneration of the coats; miliary aneurisms, such as are found in the brain, rarely develop in the cord. Hemorrhage often precedes or begins a myelitis, of which it may be the cause or the result. The clot may be absorbed, leaving a cavity as in the brain; or the broken-down tissue may become the centre of a myelitic focus. The hemorrhage is usually single, but there may be several. Multiple capillary hemorrhages occur, but usually only from asphyxia and convulsions. It is possible that some of the cases of disseminated myelitis occurring after infectious fevers start from small hemorrhages.

Diagnosis.—The points to be noted are the sudden onset without long premonitory symptoms, and the absence of fever, followed later by gradual improvement. The disease is often mistaken for acute primary myelitis, which does in fact often follow it. Meningeal hemorrhage is more painful, and there is less paralysis, more spasm, and a more complete recovery later.

Prognosis.—This is often serious as regards life, and always serious as regards health. It depends on the extent and seat of the hemorrhages. Dorsal hemorrhages are more favorable, cervical the least.

Treatment.—Absolute rest, ice-bags to the spine, and small doses of aconite given early are all that can be done, except the use of symptomatic remedies. Treatment must be applied at once. The late treatment is the same as that for myelitis.

The Caisson Disease (Diver's Paralysis).

Etiology.—The caisson disease is the name given to a more or less complete paraplegia which occurs in persons who work in caissons or diving-bells, and which is brought about by the sudden return from a condensed air to the normal atmosphere. Persons employed in caissons or bells work usually under a pressure of from one to four atmospheres, which means a pressure of from fifteen to fifty pounds to the square inch. Accidents rarely if ever occur when the pressure is not over one atmosphere, and they are also rare if the person has not been subjected to the pressure for at least an hour. Different persons vary in susceptibility to the effects of this change in the atmospheric pressure, and those unused to the work are more liable to be attacked.

The symptoms set in usually very soon after the patient has come out from the caisson, but they may be delayed for half an hour or more. They consist of intense neuralgic pains in the lower extremities, often affecting especially the joints. There is at the same time epigastric pain. Nausea and vomiting and weakness in the lower limbs, amounting in some cases to absolute paralysis, very soon appear. There may be headache, dizziness, and sometimes even coma. If the paralysis is considerable, it is usually accompanied with anæsthesia. Disturbances in the sphincters, with retention of urine and constipation, may also be present. The symptoms vary very much in severity, from pain, weakness in the legs, and nausea up to frightful neuralgic attacks and complete paralysis, motor and sensory. The upper limbs are rarely affected. In a few instances hemiplegia, however, has been observed. The disease lasts from a few hours up to several weeks. Death occurs in some of the very severe cases. The symptoms having reached their climax gradually ameliorate, and a complete cure is not infrequent. In some instances, however, the patient is left with a permanent paraplegia and the ordinary symptoms of a transverse myelitis.

Pathology.—When the patient is under atmospheric pressure in the caisson, the blood is driven from the surface of the body, and the internal viscera, including the brain and cord, are congested. The sudden change from the abnormal to normal pressure produces a rapid flow of blood from the internal organs to the periphery. The viscera not inclosed in bony cavities are enabled to relieve themselves of this congestion without much harm, but the circulation in the brain and spinal cord is less elastic; that in the spinal cord being less even than that in the brain. The result is that congestions and small hemorrhages ensue, producing a destruction of the nerve tissue. In other cases there

seems to be a blocking up of some of the small vessels, with consequent softening of different portions of the cord and to a less extent of the brain. It is supposed that one element in producing the morbid phenomena is the escape of oxygen and carbonic-acid gas from the blood into the tissues or into the blood-vessels. This mechanism, however, has not been absolutely proven. It will be seen, however, that on the whole the sudden change in atmospheric pressure leads to vascular disturbances with rupture or obliteration of blood-vessels, with consequent destruction and necrosis of tissue. Following this is a reactive inflammation producing the phenomena of an ordinary acute myelitis.

The treatment is largely prophylactic. The workmen engaged in the occupation should be carefully selected and should accustom themselves to their work. They should spend a longer time in coming out of the caisson. If symptoms supervene, it is recommended that they be put back under a slight atmospheric pressure at once until these symptoms disappear. When the disease has developed, it can only be treated by symptomatic remedies. The patient should be kept quiet and given, if necessary, hypodermics of morphine. Dr. A. H. Smith recommends the use of ergot. Later on, the various neuralgic and paralytic symptoms may be treated on the same principles as those employed in myelitis.

Spinal Hyperæmia, Acute and Chronic.

Etiology.—Acute spinal hyperæmia is produced by sexual excesses, violent physical exertion, suppression of menstrual discharges, and certain poisons like strychnine. It occurs also in the first stage of acute inflammatory diseases.

Chronic spinal hyperæmia is, so far as is absolutely known, a very rare condition. Chronic hyperæmia of the membranes may be the residuum of a meningitis or of injury, and these are probably the most common causes. As to the causes of the chronic hyperæmia of the cord substance itself independent of other diseases, we can say nothing definitely.

The symptoms of acute spinal hyperæmia are feelings of heaviness and weight in the limbs and around the loins, numbness, creeping sensations and actual neuralgic pains, weakness of the lower limbs, with twitchings of the muscles. There may be also some disturbance in the sphincters, though of this one can speak less certainly. The symptoms are nearly always confined chiefly to the lower limbs. The statement that they are increased by lying on the back and ameliorated by lying on the face is not always true, since posture, unless greatly prolonged, influences but very slightly, if at all, the circulation in the spinal cord.

The symptoms of chronic spinal hyperæmia, when this hyperæmia involves the meninges chiefly, are probably somewhat identical with those of spinal irritation. They will be described under that head.

Pathology.—The circulation of the blood in the spinal cord, as has been shown in the article on anatomy, is one which it is difficult to disturb; but which, once disturbed, is rather slow in being brought back to its normal condition. Thus violent activity of the heart and great increase in the arterial pressure, and the opposite conditions of weakened heart and lowered arterial tension, appear but little to modify the spinal functions. Hence it is unlikely that the large number of clinical symptoms that have at times been attributed to hyperæmia of the spinal cord, or rather to disturbances in the circulation of the spinal cord, have really been due to that cause.

Treatment.—The treatment of spinal hyperæmia consists in the application of cups to the back, quiet in the horizontal position, preferably upon the side or face, ice and counter-irritants to the spine, morphine and bromides internally. In the more chronic cases muriate of ammonia, iodide of potassium, and the galvanic current may be used. The use of ergot, which has been recommended, in my experience has been found of little or no value.

Spinal Anæmia.

Even less is known in regard to the etiology and symptomatology of spinal anæmia than of spinal hyperæmia. Undoubtedly severe hemorrhages or diarrhœal discharges, and an aortic obstruction which cuts off the circulation of the blood from the spinal cord, will produce a spinal anæmia, and when this is severe the functions of the cord are nearly abolished. But practically we do not know of any causes which produce an acute or chronic anæmia leading to serious and prolonged disturbances in the spinal functions, aside from diseases of the arteries of the spinal cord themselves such as occur in advanced life. In the most profound anæmias which must affect equally the spinal cord with other organs very little special disturbance of this organ can be discovered. Here, too, the supposed test of improvement on lying on the back is, in the writer's opinion, a fallacious one. It has been customary to associate with spinal anæmia a class of symptoms characterized by pains in the back of the nature of spinal irritation, weakness of the legs amounting to paraplegia—a type of symptoms that has been called spinal concussion, but it is impossible in the present stage of science to say that a spinal anæmia actually underlies and causes this condition.

INFLAMMATION OF THE SPINAL MEMBRANES (SPINAL MENINGITIS).

The meningeal inflammations are:

External meningitis. Internal meningitis.	Affecting the dura mater.
Leptomeningitis.	Affecting the pia mater.
Hypertrophic pachymeningitis.	Affecting both membranes.

External Meningitis, Pachymeningitis Externa ("Compression Myelitis").

Etiology.—The disease is rare, and always occurs secondarily to some other morbid process. This process is in most cases tuberculosis and caries of the vertebræ. Other causes are suppurative inflammation in the neighborhood of the vertebræ, psoas abscess, purulent pleurisy, peritonitis, and puerperal pyæmia. When the disease arises from inflammations in the visceral cavities, it is thought to be caused by an ascending neuritis.

The Symptoms.—The symptoms are those of irritation of the motor and sensory roots; later, compression of them and of the spinal cord, local pain in the back, radiating pains, tenderness, hyperæsthesia, twitching, paresis, paraplegia, exaggeration of reflexes, and involvement of the sphincters. Anæsthesia occurs in severe forms.

The disease, when chronic, may extend to the other membrane and cord, causing what is termed "compression myelitis."

Pathological Anatomy.—The inflammation, if acute, is generally a fibro-purulent one, this being the form usually caused by vertebral caries. The dura mater is covered by a layer of caseous, semi-solid matter, often very thick and most extensive posteriorly. It involves the dura vertically for several inches. In chronic forms the deposit is made up of connective tissue and the cord is compressed. In purely suppurative forms the cellular tissue outside the dura is infiltrated with pus throughout a great part of the canal.

The *diagnosis* is based on the presence of the primary local disease, the kyphosis, the radiating pains, and tenderness, and by the combination of motor and sensory irritation and paralysis.

The *prognosis* is generally bad because the original disease is a serious one.

The *treatment* consists in attention to the local caries or inflammatory focus. It is therefore purely surgical.

Internal Meningitis, Pachymeningitis Interna, Hemorrhagic and Hypertrophic.

Inflammation of the inner surface of the dura mater occurs in two forms—the hemorrhagic and the hypertrophic. As the latter is generally but a sequel of the former, I shall describe the two together under the head of *hypertrophic pachymeningitis.*

Etiology.—The disease occurs almost always in adult life. A few cases have been reported in children (Gibney). It usually affects males. Syphilis, alcoholism, exposure, and trauma are exciting causes.

Symptoms.—The disease begins gradually with symptoms of irritation (irritative stage). The patient suffers from pain and stiffness in the neck. The pains radiate up to the occiput and down the back; numbness, prickling, and pain are felt in the arms, more in one than the other. The pains exacerbate and are worse at night. Stiffness and cramps may affect the arms. Nausea and vomiting sometimes occur.

After five or six months, symptoms of paralysis appear (paralytic stage). The arms are first affected. They become weak, atrophy occurs, associated with contractures and rigidity. There is still pain, and in addition anæsthesia, hyperæsthesia, and trophic changes. Later, paraplegia, with rigidity, exaggerated reflexes, and spinal epilepsy, develop. The patient becomes weaker, and finally dies of exhaustion or from some intercurrent trouble. The disease sometimes takes what is called the *peripheral type.* Then the symptoms are more localized in the extremities. Usually it is of the *cervical type* and presents symptoms as described above. In either form the disease is a chronic and painful one.

Pathology.—The disease starts as a hemorrhage upon the surface of the dura. This leads to a chronic inflammatory process, new hemorrhages occur, and a fresh inflammatory deposit is made until the cord is finally encircled and compressed by a dense connective-tissue mass, which involves the pia and to some extent the cord substance. The process is analogous to that of cerebral pachymeningitis hæmorrhagica. The cervical region is usually attacked.

Prognosis.—A few cases have been reported practically cured. More cases eventually die. Sometimes, however, the disease comes to a standstill for a long time.

Diagnosis.—This must be made from tumor, myelitis, Pott's disease, wry-neck, and progressive muscular atrophy. The history of injury, the slow progressive course, and the localization of the symptoms, their bilateral character and the pain, give the most help. It is not always possible to exclude a tumor.

Treatment.—The not rare syphilitic origin of the disease must be borne in mind. Counter-irritants, electricity, hydrotherapy, and symptomatic remedies for the pain and spasms are indicated. It is possible that surgery may help these cases.

Acute Spinal Leptomeningitis (Inflammation of the Pia Mater of the Spinal Cord).

Etiology.—This is a rare disease, occurring alone, but is common in connection with disease of the cerebral pia mater.

Children are oftenest affected; and among adults, males. Alcoholism predisposes to it. The disease is always secondary to an infection or a traumatism. The infections are tubercle, syphilis, typhoid fever, and septicæmia. Extension of inflammation from neighboring parts and surgical operations are occasional

causes. The cases attributed to rheumatism, exposure, insolation, are in reality due to some infection, and the virus of cerebrospinal fever sometimes attacks the cord alone.

Symptoms.—The disease begins with pain in the back, radiating along the nerves. There is usually a chill and some fever. The pulse may be fast or slow. The pain increases, and is accompanied with dorsal tenderness and rigidity of the muscles of the back, amounting sometimes to opisthotonus. The skin is very hyperæsthetic and the reflexes are at first increased. There is constipation, and sometimes retention of urine. After a time symptoms of paralysis come on, with anæsthesia and retention of urine. The patient becomes weaker, bed-sores may form, and death from exhaustion follow. The disease lasts from a few days to several weeks.

The dominant symptoms in the first stage are those of irritation, viz.:

Pain in back and along the nerves, hyperæsthesia, muscular spasm. In the second stage, paralysis, atrophy, and anæsthesia.

In the *tubercular* form of meningitis the symptoms come on more slowly. In *septic* meningitis the symptoms are of the severe and typical kind described. In meningitis from other infections the symptoms are not so severe as a rule.

Pathological Anatomy.—Acute leptomeningitis shows a somewhat different exudate according to the nature of the infecting micro-organisms. The common form is the suppurative exudate which may be due to the streptococcus pyogenes and other pyogenic micro-organisms, and to the pneumococcus. The purulent matter is usually distributed along the whole length of the cord, but more posteriorly and often more in its lower portion. It may also be localized chiefly at certain levels. The spinal fluid is increased in amount. The arachnoid, the inner surface of the dura, and the tissue of the cord itself are usually involved. If the disease lasts several weeks, the purulent matter is absorbed in part and an increase in connective tissue takes place, binding the dura, arachnoid, and pia to the cord. The nerve roots are surrounded and compressed by the inflammatory product.

In tubercular meningitis there is less exudate. It is more of a fibrinous character, and grayish in appearance. Tubercle granulations are seen distributed over the pia, arachnoid, and inner surface of the dura. Simple exudative meningitis rarely occurs. The inflammatory process often ends in a production of new connective tissue and sometimes the establishment of a chronic leptomeningitis.

The *diagnosis* must be made from myelitis, tetanus, rabies, rheumatism of the dorsal muscles, gonorrhœal rheumatism, and strychnine-poisoning. In myelitis there is relatively little pain

and much paralysis; in tetanus there is trismus, fever is absent, and there is a history of trauma.

Tubercular meningitis comes on more slowly, is rarely spinal alone, and there may be evidence of local tuberculosis elsewhere.

The *prognosis* is not good, but is especially bad in tubercular meningitis and in cases with high fever, severe pains, and early paralysis. Chronic meningitis sometimes remains after the acute symptoms subside.

Treatment.—This consists first in perfect rest and quiet; leeches should be applied along the spine, then hot poultices or ice-bags; opium is to be given for relief of pain; mercurial purges and small doses of iodide of potassium or sulphate of magnesium may be given at short intervals. Later, blisters and counter-irritation and lukewarm baths are indicated.

Chronic Leptomeningitis and Meningomyelitis.

Etiology.—This disease, which used to be often diagnosticated, is now believed to be rare, and always secondary to an acute process or to syphilis and perhaps chronic alcoholism. It occurs oftenest in adults and in males. Trauma, and especially concussion of the spine, used to be thought a frequent cause, but in most of such cases the trouble is simply a hyperæmia or else neuralgic and functional.

The *symptoms* gradually develop after an acute meningitis or an injury, and they are the same in character as those of the acute process. There is pain in the back, increased on movement and radiating about the trunk and down the limbs; tenderness along the spine, stiffness of the back, twitching and spasms in the limbs with some weakness, and later some paralysis, wasting, and anæsthesia, with weakness of the bladder. These paralytic symptoms, if severe, however, mark an invasion of the spinal cord. Cutaneous eruptions, such as herpes, may appear. The symptoms run an irregular course, with periods of improvement. They always become less when the patient rests.

Pathological Anatomy.—The inflammation consists of a proliferation of connective tissue (productive inflammation of Delafield). The result is a thickening and opacity of the pia mater and arachnoid. The dura mater may be involved, but only in severe cases. The process may invade the spinal cord, causing an "annular" or ringlike sclerosis, from which the disease extends into the cord in wedge-shape masses at various parts, causing eventually an involvement of fibre-systems and secondary degenerations up and down. This somewhat rare terminal condition is called meningo-myelitis.

The *diagnosis* must be made from spinal irritation, locomo-

tor ataxia, myelitis, vertebral caries, and torticollis. In spinal irritation there are not the rigidity, severe radiating pains, twitchings, atrophy, or paralysis; and neurasthenia or hysteria exists. In locomotor ataxia the knee jerk is lost, there is ataxia, and there is little paralysis, nor is there local tenderness over the spine.

In vertebral caries the pain and tenderness are much more localized, and there is spasmodic fixation of the trunk. The pain is more continuous and dull, and is increased by lateral pressure and lessened by extension. There is usually also some deformity. If compression occurs there is exaggeration of the reflexes and paraplegia, without much anæsthesia. After all, however, with Pott's disease there may be a local meningitis.

Treatment.—As chronic meningitis is usually the product of syphilis or the relic of traumatism or of an acute process, the indications for treatment are simple. Rest is the essential thing. With this could be combined the systematic and persistent use of counter-irritants. The hot iron is usually best, because its wounds heal so quickly. Cupping is also useful if done vigorously and often. Internally, iodide of potassium, small doses of bichloride of mercury, nitroglycerin, and digitalis may be given; also the salicylates and ergot. Both the latter drugs should be employed in large doses, if at all. Electricity in the form of galvanism and cocaine by local injection, or cataphoresis, may be tried.

CHAPTER XII.

MYELITIS—INFLAMMATION OF THE SPINAL CORD.

MYELITIS is an inflammation of the spinal cord. It may be *acute*, *subacute*, or *chronic*.

It may affect the anterior horns chiefly, when it is called *anterior poliomyelitis;* or both the gray and white matter, when it is called *diffuse myelitis* and *transverse myelitis*. The forms are still further divided, in accordance with their location, into *annular* or *marginal* myelitis where the inflammation involves the periphery of the cord; *disseminated myelitis* where it occurs isolated in different parts of the cord; *periependymal myelitis* where it surrounds the central canal. Myelitis is given different names also in accordance with its cause. Thus we have *hemorrhagic myelitis*, a form in which the initial process is due to or associated with a hemorrhage; *compression myelitis*, due to vertebral caries, *septic* or *purulent* myelitis or abscess of the cord; and *tubercular* and *syphilitic* myelitis.

For practical purposes the following classification is sufficient:

Acute myelitis—	
With exudation and necrosis.	1. Anterior poliomyelitis. 2. Transverse myelitis.
With suppuration.	3. Abscess of cord.
Chronic myelitis—	
With necrosis and proliferation.	4. Chronic anterior poliomyelitis 5. Transverse myelitis.
Rare forms.	6. Marginal myelitis. 7. Periependymal myelitis. 8. Disseminated myelitis. 9. Compression myelitis.

ACUTE TRANSVERSE MYELITIS (ACUTE SOFTENING OF THE SPINAL CORD).

Definition.—Acute myelitis, so called, is generally an acute softening of certain parts of the cord, with secondary inflammatory reaction. It is in extremely rare cases a primary exudative and necrotic inflammation. In other rare cases the inflammation is suppurative, and we then have *spinal abscess*.

Forms.—The common form is one that involves one or two

inches vertically of the spinal cord, and is hence called *acute transverse myelitis.* Since both gray and white matter are involved, it is also called a diffuse myelitis.

Acute transverse myelitis (softening) *is the common type.*

An acute *disseminated myelitis* sometimes is met with. Other rarer types are named, in accordance with their location: marginal, central, or periependymal. There is also an acute meningomyelitis, the result usually of a meningitis.

Etiology.—Predisposing causes are a neuropathic constitution, the male sex, early adult life, occupations calling for exposure, and muscular strain. The exciting causes are exposure to cold, blows, falls, fractures, strains, extension from neighboring organs, syphilis, infective fevers, and septic infection. *Injury* is the most frequent cause and *syphilis* next. But most of these causes produce primarily mechanical destruction, hemorrhages or thromboses, and the inflammation is secondary.

Symptoms.—Prodromal symptoms are rarely present, but there may be slight paræsthesiæ or pain in the back and limbs. Sometimes there is a chill, and in a few instances convulsions have been noticed.

The *initial* symptoms consist of feelings of numbness, usually in the feet and legs, which seem heavy and weak. Some pain may be felt about the back. The patient soon finds that he cannot walk easily and that he moves his legs with an effort. In a few hours a paraplegia with anæsthesia has developed, and if the lesion is in the cervical cord the arms are paralyzed also. All these symptoms may come on in the day-time or during sleep. Retention or incontinence of urine and constipation are early symptoms. There may be some fever.

Symptoms of the Attack.—If the patient is now examined, it will be found that he cannot walk or stand, but can move his legs a little. He complains of a sensation like a band around his waist or at the level of the spinal lesion (girdle-symptom). His legs feel numb and heavy, but there is little pain and no tenderness. Anæsthesia to touch, pain, and temperature exist in various degrees on the limbs as high up as the lesion. The anæsthesia, if not total, is greatest to touch, next to temperature, and least to pain. He has vesical anæsthesia, the urine is retained and he has to have it drawn. The bowels are constipated, but if enemata are given the fæces may pass away without his knowledge, owing to rectal anæsthesia. If the lesion is lumbar there is abolition of the sexual power; but if dorsal or cervical, strong erections may occur without the patient's feeling them. When the lesion is above the lumbar cord, also, the bladder may automatically and forcibly contract and expel the urine. In time the bowels regain some power. The paralysis in the limbs affects the

flexors of the feet and legs more than the extensors. The patient can push down his limbs better than he can draw them up.

The temperature of the limbs for a few days is raised, but after this it falls a few degrees below normal. The skin becomes rough, cold, congested; and excessive perspiration may take place. The general bodily temperature is usually normal throughout the disease, but in some cases a fever develops of 102° to 104° and continues. The prognosis is then bad.

Bed-sores may develop early, within a few days or weeks. They appear oftenest upon the buttocks and heels, and are due to trophic disturbance, combined with pressure and pyogenic infection of the parts.

If the lesion is lumbar, the tendon and skin reflexes are much weakened and the paralysis is somewhat flaccid. The muscles also tend to waste and show degenerative reactions. If the lesion is dorsal, as is more often the case, the reflexes are present, and after a time become exaggerated; contractures and spasms develop; the legs may become drawn up and deformities produced. If the lesion is so complete as to entirely cut across the cord, there may still be some excessive muscular tension, but the reflexes will be abolished (Bastian). When the cervical region is attacked the arms are involved as well as the legs, and generally to a severer extent. There may now be also unequal dilatation of the pupils from involvement of the cilio-spinal centre; and optic neuritis from some cause has been known to occur. In extensive involvement of the upper part of the cord there may be paralysis of the intercostal muscles and disturbance of the heart's action.

The disease having in a few days reached its height, usually remains stationary for a few weeks, and then, should the patient live, improvement slowly sets in. In some cases evidences of extension upward or downward occur (ascending or descending myelitis); the symptoms become more severe, and in a few weeks, or oftener months, death occurs.

As improvement begins, a return of sensation is first noticed (one to six months); this is followed by return of more or less motion (six to eighteen months). Spasms and contractures now develop, owing to a descending degeneration. A certain amount of ataxia from ascending degeneration, with a little anæsthesia of the skin, may remain, so that if the patient has sufficient motor power to walk somewhat, he presents many features of "ataxic paraplegia" (see p. 249).

Some improvement may be expected for from one to two years. A few cases get almost entirely well. The majority become more or less paraplegic and bed-ridden, in which condition they are regarded as cases of chronic myelitis.

Pathological Anatomy.—The early changes found in acute

myelitis are those of inflammation, hemorrhagic extravasation, and softening. Often it is impossible to say whether the primary process was inflammatory or due to a hemorrhage or softening.

Macroscopically, the cord at the affected part appears soft, swollen, and either red and hyperæmic or pale and anæmic. In rare cases no change is apparent to the naked eye. In later stages the parts are white or gray, shrunken, and hard. The cord may be reduced to a thin shred. The meninges about the affected parts are often thickened, inflamed, and adherent.

Microscopically, if the process is primarily inflammatory we find intense congestion, distended blood-vessels, emigrated white-blood cells in great number, especially in the perivascular spaces, swollen axis cylinders and œdematous swelling of the myelin sheath, red-blood cells, cells filled with fat granules known as compound granular corpuscles, or Gluge's corpuscles. These are leucocytes which have taken up fat granules. A peculiar form of cell, stellate in shape, known as spider cells or Deiter's cells, may be seen. They are proliferated neuroglia cells. The nerve cells lose their granular or reticular appearance, become homogeneous, swell up, fat granules appear in them, and a peculiar coagulation process attacks the cell and body. The cell processes retract, become thick, and finally drop off. Vacuoles are sometimes seen in the nerve cells; but these are very rare if the tissue is properly preserved. Besides the above evidences of vascular and connective-tissue activity and of cell destruction, one sees granular matter, broken-down nerve fibres, and other evidences of nerve disintegration. Pigment or extravasations of blood, and in later stages bodies resembling starch granules (corpora amylacea), may be seen. They are probably modifications of the myelin substance. If the lesion is primarily softening there may be much congestion, but the blood-vessels are less distended and fewer white cells are found in the perivascular spaces. The connective-tissue cells are less numerous. The nerve cells are swollen, glassy, and stain badly. There is a great deal of granular matter and nerve detritus.

It is thought that in inflammatory swelling of the nerve cells they take up the carmine stain, but in degenerative changes they do not.*

* In the different stages of acute degeneration, the nerve cells, particularly those of the anterior horns, show various appearances. These have been described by Friedmann as—1, homogeneous swelling, hyaline swelling; 2, sclerosis; 3, simple atrophy; 4, fatty and molecular decomposition; 5, cloudy swelling. The degeneration begins in the cell body and then involves the processes and the nucleus; finally the whole cell is involved. Nerve cells do not often undergo the acute general death called coagulation necrosis (Friedmann), though the process of sclerosis so called resembles it somewhat.

The connective-tissue changes are most noticeable in the white matter. The axis cylinder and myelin sheath are here often so disintegrated that in thin sections they drop out, leaving holes in the section and giving it a vacuolated appearance. The lesion, whether destructive or inflammatory, may extend up or down the cord, often in the course of certain tracts. Sometimes a destructive process extends a long way through the central part of the cord (perforating necrosis).

After three or four weeks, if the patient lives, the process of absorption and cicatrization begins, and secondary degenerations occur. The granular and fatty matters and leucocytes disappear, the blood-vessels are thick-walled and distended but less numerous, connective tissue gradually takes the place of the destroyed nerve cells and fibres. The axis cylinder is the last to be destroyed and has the greatest power of recuperation. The nerve cell, if once destroyed, is never developed again. The cord at the point or points inflamed or softened becomes reduced to a mass of connective tissue containing, perhaps, a few nerve fibres and cells. In this stage the condition is one that is known as chronic myelitis. In some cases a cyst is formed in the affected region.

In fatal cases the inflammation and softening continue; fresh areas of cord are involved, much meningeal exudation takes place and finally death occurs. The process may in very rare cases be still more acute, suppuration and abscess occur, and here death also rapidly ensues.

The inflammatory and softening processes above referred to are described in accordance with their appearance as red softening, yellow softening, white softening. A form of so-called inflammation known as inflammatory œdema is also described. It is an abortive inflammation, "a lymphatic congestion," analogous to vascular congestion.

The *diagnosis* of acute myelitis must be made from hemorrhage, acute embolic or thrombolic softening, acute ascending paralysis, multiple neuritis, meningitis, and meningeal hemorrhage, and hysterical or functional paralysis. Spinal hemorrhage comes on suddenly and is usually not attended with fever. If meningeal, it is attended with pain. Acute softening cannot be distinguished from acute myelitis, of which it is probably the starting-point in many cases. In acute ascending paralysis the disease is progressive, there is no involvement of sensation, no atrophy, and little change in the electric irritability. In multiple neuritis the onset is slower, there is more pain and local tenderness and sensory disturbance, and the bladder and rectum are rarely involved. In meningitis there is pain and tenderness in the back and limbs, rigidity, cramps, a little paralysis,

and no bladder trouble. In hysterical paraplegia there are no marked atrophic changes, but little spasm or rigidity, no electrical changes, and the stigmata of hysteria may be found. The sensory disturbances are variable and somewhat characteristic.

The diagnosis of the *location of the lesion* is made by studying the height of the anæsthesia, the skin reflexes (see p. 190), and the distribution and extent of the paralysis. Lesions of the lumbar region involving the gray matter cause very complete paraplegia with sphincter troubles and degenerative electrical reactions. Lesions in the dorsal cord cause a less complete paraplegia, but, owing to the secondary descending degenerations of the lateral column, rigidity, exaggeration of the deep reflexes, and contractures occur. Lesions of the cervical cord cause paralysis of the arms, with degenerative reactions in the muscles. The paraplegia is spastic and there is not much muscular wasting. If the lesion cuts off the cord entirely, the limbs are paralyzed and may be somewhat rigid, but the deep reflexes are absent.

Prognosis.—The prognosis is worse the more complete and extensive the paralysis. It is worse in serious motor paralyses than when sensation is chiefly involved.

It is best in dorsal myelitis and worst usually in cervical myelitis, other things being equal. Bed-sores and slight fever are unfavorable signs, so also is severe involvement of the bladder and rectum. Recovery of sensation gives good hope of recovery of some motion. Total absence of recovery of sensation and motion after six months is very unfavorable. Improvement may be expected up to eighteen months after the onset, and in some cases even longer. In compression myelitis there is more chance of recovery than in the other forms. The previous existence of syphilis does not affect the prognosis of cases of true myelitis.

Treatment.—In the attack, the patient must be put to bed, leeches or wet cups should be applied to the spine, diaphoresis should be promoted, small doses of aconite and nitroglycerin should be given, and a calomel purge administered. The bladder should be watched. After a week, moderate doses of iodide of potassium should be given. After about three weeks, if there is no fever, electricity may be applied carefully and strychnia administered. Bed-sores should be guarded against by the use of water beds or cushions, absorbent cotton, bathing the parts with alcohol and weak solutions of tannin. Infusion of buchu, boracic acid, and tincture of hyoscyamus will often help the bladder disturbance. After acute symptoms subside, tonics such as arsenic, iron, and strychnia in small doses may be given. Suspension may be tried carefully; mechanical appliances may be used to help the rigid limbs; lukewarm baths, douches, and massage are helpful to some extent.

Chronic Myelitis (including Transverse, Diffuse, Disseminated, and Compression Myelitis).

Chronic myelitis is the name given to a disease characterized by a chronic inflammation of the spinal cord and to the chronic reparative processes which follow acute inflammation, injury, and softening. Chronic myelitis is usually a mixture of inflammatory, reparative, and necrotic processes.

Forms.—Different names are given to chronic myelitis in accordance with the part of the cord affected. Usually the disease affects only certain levels, and then it is called *transverse myelitis.* More rarely it is *diffuse* or *disseminated*, *central* or *marginal.* When caused by pressure from vertebral disease it is called *compression myelitis.*

Etiology.—The disease may be either *primary* or *secondary.* The *primary* form is much the rarer. It occurs chiefly in adults and in early and middle life, and much oftener in males. Exposure, shocks, infectious fevers, lead, and syphilis are the chief causes. Syphilis causes it by producing arterial disease and by setting up a specific inflammatory infiltration. *Secondary* chronic myelitis is the more common form. It is really only the later stage of acute myelitis, softening, hemorrhage, or injury.

Meningitis may extend and cause a meningo-myelitis. A neuritis may possibly ascend and cause myelitis, but such cases, if they occur, are very rare. Compression myelitis is usually a slowly destructive, not an inflammatory process, and it begins as a meningitis of the dura mater.

Symptoms.—When the disease begins primarily as a chronic affection the symptoms are as follows:

The patient notices that his legs are heavy and get easily tired; prickling and numb sensations are felt in the feet; occasionally a little pain develops in the back or there is a sense of constriction about the trunk. The legs feel stiff, and tests often show that the reflexes are exaggerated. There is but little wasting of them, however. The sexual power declines; the bladder gives some trouble, there being a tendency to retention; the bowels are constipated. After a few weeks or months there is a partial paraplegia, with rigidity of the limbs and exaggerated reflexes. Some anæsthesia exists, and occasional pain, which is not severe and is felt more in the back than the legs.

The muscles have now wasted somewhat, but show no decided changes to the electrical current. The bladder becomes more involved, the urine has to be drawn, it is often alkaline, and unless care is taken cystitis develops. The patient is still able to walk, but he does so with a stiff, shuffling gait which is characteristic

(Fig. 93). The disease may show signs of slowly extending up and down, more often up. The arms become involved; weakness and stiffness, with some wasting, anæsthesia, and pain, develop, or the disease may cease its progress and the patient remain partly paralyzed for years. The general health during the course of the disease deteriorates slowly; the patients often become anæmic and have an unhealthy pallor. Eventually the paraplegia becomes complete, the patient is bed-ridden, the legs are atrophied, contractured, and rigid, with more or less anæsthesia. Cystitis and nephritis develop, or the patient dies from this or from intercurrent disease.

Fig. 93.—Attitude in Chronic Myelitis.

Chronic secondary myelitis, which is the form commonly seen, presents eventually much the same picture as that just described. In this type, however, the symptoms are worse at first, then improve or regress, then become stationary, and finally grow worse.

Symptoms of the Different Forms.—The usual type of chronic myelitis is the transverse dorsal or dorso-lumbar, and this gives symptoms as above described. If the lumbar region is affected there is more paraplegia, wasting, and involvement of organic centres. If the myelitis is cervical the arms are involved, there may be pupillary changes, and the respiratory muscles are partly paralyzed; the paraplegia is not so complete and the disturbances of sensation are likely to be more varied.

Compression myelitis, so called, is usually only a compression atrophy. It is due, as a rule, to vertebral caries, but its cause may be a spinal tumor, aneurism, and pachymeningitis. Compression myelitis is distinguished from other forms by its slow onset and the presence at first of irritative or "root" symptoms. The patient suffers from pain and tenderness localized at a certain point in the spine. The pain radiates about the trunk or down the limbs and is increased on movements. At about the same time some motor weakness develops, usually in the form of paraplegia. The muscles waste but slightly. The reflexes are exaggerated; twitchings, spasms, and contractures finally oc-

cur, and there is finally developed a spastic paraplegia or quadruplegia. With this there is usually some anæsthesia, though it is not complete. The disease is oftenest in the dorsal or lower cervical region, and hence the sphincters escape until late. Locally, evidences of spine disease appear early in the form of a kyphosis.

A *central* or *periependymal myelitis* can rarely be recognized with certainty. It produces less pain and irritation, but leads to muscular atrophy, disorders of sensations such as thermo-anæsthesia, disturbance of vasomotor and secretory nerves and visceral centres.

Pathology.—The pia mater is thickened over the affected region and often throughout the cord. The cord itself has a gray, discolored look at the affected level, and is usually shrunken or distorted and hard to the touch. In severe cases of secondary character it is reduced to a small size, and the membranes about it are thick and inflamed. In transverse myelitis a vertical area of only two or three inches is involved. The microscope shows that the prominent changes are loss of nerve structure, great increase of connective tissue, and increase in the number of vessels which often have thickened walls. In the more seriously diseased part little is seen but connective tissue. In parts less diseased some nerve fibres are seen, many having evidences of partial disintegration. There is also a good deal of amorphous material studded with nuclei. Stellate cells, granule cells, and nerve cells in various stages of degeneration are present. In the parts less affected the signs of congestion and vascular irritation are more pronounced.

Diagnosis.—This must be made from progressive muscular atrophy and amyotrophic lateral sclerosis, pachymeningitis and spinal tumor; from locomotor ataxia and from brain palsies.

In progressive muscular atrophy there is a peculiar atrophy without involvement of the sphincters or sensory disturbance. In pachymeningitis there is often a history of an injury; there is more pain in the back and a more marked anæsthesia. The sphincters are not involved. Pachymeningitis is also usually located in the cervical region. Tumors usually cause much more pain; the symptoms come on slowly and are more definitely localized. A spastic paraplegia occurs from brain disease and as a functional trouble. In either case there are no trophic or sensory troubles, nor is there involvement of the sphincters. In locomotor ataxia there is no great degree of motor paralysis, and there are peculiar ataxic and sensory disturbances. Paralysis from brain disease is almost always unilateral, painless, spastic, and free from disturbance of the visceral centres.

Prognosis.—Inflammatory processes have a tendency to cease when their reparatory and eliminative work is done. Chronic

myelitis, however, is often, as has been stated, a destructive process due to some defect in vascular supply or to some mechanical irritation. Besides this, in the spinal cord secondary degenerations set in as soon as certain tracts are interfered with. Hence chronic myelitis, after a period of improvement, generally progresses, and the prognosis is not very favorable. Still, patients may live from five to twenty-five years. Dorsal myelitis is the most favorable form; compression myelitis from caries can also often be successfully treated. Those forms which come on rather rapidly are more likely to cease progressing (Gowers). Serious involvement of the bladder is a bad sign, and naturally the prognosis is worse the more complete the paralysis.

Treatment.—In the earlier and progressive stage of chronic myelitis rest is imperative. The patient should lie down much of the time. Counter-irritation in the form of fly blisters, the cautery, or setons should be applied, and if no improvement results wet or dry cups used. The descending galvanic current along the spine should be tried; faradism and massage being used upon the limbs. Cold baths and cold applications must be prescribed carefully if at all. Lukewarm baths 90° to 98 °F. or half-baths with friction at 70° to 80° F. are more likely to be useful, but even these must be tried cautiously. The first baths should last not over five minutes and should be repeated only three or four times weekly. In later paraplegic and bed-ridden stages electrical and hydro-therapeutic applications should be followed up patiently and persistently. The patient now may be allowed to remain and exercise in the lukewarm bath for some time.

Internally, iodide of potassium and mercury should be first given. After thorough trial with these remedies for six or eight weeks, the patient should be given courses of treatment with arsenic, nitrate of silver, phosphorus, and perhaps the chloride of gold. Pills of arsenite of soda, gr. $\frac{1}{30}$, may be administered three or four times daily for two months; if benefit ensues, the remedy should be resumed after an intermission of three weeks. Phosphorus is best given in the form of Thompson's solution or Sayres' elixir in doses of gr. $\frac{1}{60}$ ter in die increased to gr. $\frac{1}{10}$. The remedy should be suspended for three days at the end of each ten days. Silver is given usually in the form of the nitrate (dose, gr. $\frac{1}{6}$ to $\frac{1}{3}$). Not more than one drachm should be given without a three-months intermission. Some assert that the hypophosphite of silver and sodium, or the albuminate, are surer preparations. I do not advise the use of ergot. Strychnia in small doses sometimes is useful. For the bladder troubles, the internal use of boracic acid, buchu, santalwood, and similar drugs are useful. Mechanical and surgical measures may be of some help. In very

rare cases suspension does good, but it may do harm. Tenotomy is justifiable for the purpose of straightening contracture limbs. In compression myelitis suspension on an inclined plane and the plaster jacket or other support are indicated. Cases have been reported in which surgical operations for the relief of a supposed tumor have cured compression myelitis from Pott's disease. Sea voyages are often useful and are preferable to mountain climates. Rest, quiet, fresh air, and a very regular life are the essentials in all climates.

Acute Anterior Poliomyelitis (Infantile Spinal Paralysis, Acute Atrophic Paralysis).

Anterior poliomyelitis is a disease of the spinal cord characterized by a motor paralysis of rapid onset, followed by muscular wasting, without sensory symptoms. It occurs at all ages, but vastly oftener in infancy; hence it is often called infantile spinal palsy.

Etiology.—The average age at the time of attack is two years. Most cases occur under ten, and four-fifths of these occur under three. It may be congential, *i.e.*, occur in intra-uterine life (Sinkler), and it may occur as late as sixty. Most adult cases occur under the age of thirty.*

Rather more of the infantile cases occur in boys, and most of the adult cases occur in males.

Race and climate afford no exemption so far as is known.

The great majority of cases occur during the hot months of summer (Sinkler). Nearly eighty per cent (78.8) occur between June and September, inclusive.

Heredity has an influence in only one or two per cent of cases.

Over-exercise and chilling of the body when heated are occasional causes. Infectious fevers (oftenest measles) precede the attack in about seven per cent (Sinkler). The disease has been reported to have occurred as an epidemic (Cordier). Dentition is rarely an exciting cause, as used to be supposed. Injuries and falls in a few instances appear to be the cause of the disease. The fact that the child is just beginning to walk at the period when most susceptible to poliomyelitis must be considered of importance, since the new movements call for an unusual activity of the spinal centres.

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* Among 50 personal cases, 39 were in children. Of the latter there were 24 males, 15 females. Ages: Under six months, 1; half to one year, 8; one to two years, 10; two to three years, 5; three to four years, 3; four to six years, 3; six to eight years, 3. Final result was palsy, chiefly in right leg, in 14; chiefly in left leg, in 5; in both legs, 5; in right arm, 4; all four extremities, 1; the remainder in various combinations. Disease followed scarlatina in 1, pertussis in 2, cholera infantum in 1, some "fever" in nearly all cases.

To sum up, *age*, *season*, and *infectious diseases* are the three most important etiological factors.

Symptoms.—There are rarely any premonitory symptoms. The patient is taken with a slight fever, 100° to 102°, accompanied with vomiting, diarrhœa, or convulsions. In a few hours or a day paralysis develops; sometimes the paralysis is as much as a week in developing ("subacute form"). The fever lasts from one to three or four days. The paralysis reaches rapidly its height, then remains stationary for a time; then improvement sets in, which reaches a certain point and then stops.

We have consequently:

1. A stage of invasion—a few hours or a week.
2. A stationary period—one to six weeks, usually two weeks.
3. A stage of improvement—six months to a year.
4. A chronic stage.

1. The stage of invasion may be so sudden as to suggest hemorrhage. Sometimes the child, after a restless, feverish night, wakes in the morning paralyzed. Usually the initiatory symptoms last less than a day. With or before the paralysis there is a fever of 100° to 102° F., vomiting, delirium, and, much less often, convulsions, which are not severe. After the general disturbance subsides there may be some pain in the back and limbs for a few days, and in rare cases the bladder is involved so that there is retention of urine. But the dominant symptom is motor paralysis.

The paralysis is oftenest paraplegia, next one leg, next the arms and legs, and after this various combinations. The eye muscles, laryngeal and respiratory muscles, always escape in infants. In older persons the facial nerve may be involved. In certain peculiar cases the cranial nerve nuclei are attacked, in association with the anterior horns. When the eye muscle nuclei are involved it is called "polio-encephalitis superior;" when cranial nerve nuclei lower down are involved it is called "polio-encephalitis inferior."

2. The paralysis reaches its height in from one to four days.

It remains at its height for from one to six weeks, and then improvement gradually sets in. In two or three weeks a wasting of the paralyzed limb may be noticed. It is flabby, its temperature lowered, and the reflexes are gone. Slight tenderness may be present, but there is no anæsthesia.

3. The stage of regression lasts for from one to six months. There is gradual improvement of the paralysis, beginning in the limbs least affected. This continues until the paralysis has left all but one or two limbs. As a rule, it is the legs alone that are finally left paralyzed. In a quarter of the cases both legs, and in half of the cases one leg, oftener the right, remain affected. The

muscles waste and show reaction of degeneration, viz., loss of faradic irritability, retention but lessening of galvanic irritability, sluggish contractions, and sometimes polar changes. In the leg the anterior tibial group of muscles is oftenest affected, in the arm the deltoid and shoulder group. The atrophy, having reached a certain grade, finally ceases, and then a slight improvement may set in. After the end of a year not much further spontaneous improvement can be expected.

4. The temperature of the affected limb is lowered several degrees, the skin has a reddish-purplish, mottled look. The bones as well as muscles of the affected limbs do not grow as fast as those of the healthy limb. Hence in time the foot becomes smaller and the leg shorter. Owing to the contraction of unop-

Fig. 94.

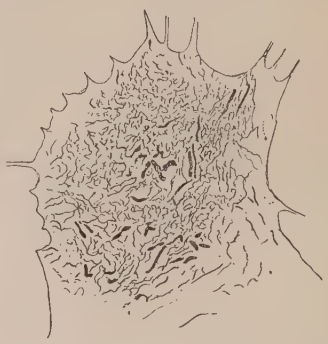


Fig. 95.

Fig. 94.—Anterior Poliomyelitis in Lumbar Segment, showing intense (Demaschino).

Fig. 95.—Same, Much Enlarged, showing congestion.

posed muscles deformities occur. The most frequent are talipes equinus, talipes vulgus and varus. Deformities of the knees, contraction of the plantar fascia, lateral curvature of the spine, also take place. The general health of the patient is usually good.

Pathology.—The disease is an acute exudative inflammation with destruction of tissue, but without suppuration. It affects chiefly the anterior cornua, especially of the lumbar and cervical enlargements (Figs. 94, 95). It is not, as a rule, diffuse, but often the brunt of the trouble is felt only by certain cell groups. These are destroyed, and after a time connective tissue takes their place. In rare cases the larger part of the central gray and some of the white matter are involved. Later a certain amount of sclerosis occurs in the lateral columns. The anterior roots and motor nerves atrophy. The muscle tissue also wastes, and its place is supplied by connective tissue.

Diagnosis.—The disease must be distinguished from multiple neuritis, spinal hemorrhage, cerebral palsies, birth palsies, and progressive muscular atrophy. The diagnosis can be easily made in almost all cases by remembering these facts:

1. The age of the patient.
2. The abrupt onset and rapid development of extreme paralysis.
3. The tendency to improve.
4. The absence of anæsthesia, bladder or rectal symptoms, rigidity, and pain.
5. The electrical reactions.
6. The arrest of growth of the limb. Multiple neuritis and progressive muscular atrophy rarely occur in children. Myelitis and hemorrhage are usually accompanied by sensory disorders, bed-sores, and bladder troubles; cerebral palsies are usually unilateral and accompanied with symptoms of stiffness and exaggeration of reflexes.

Prognosis.—The patient rarely dies, either from the disease or its sequelæ. He always improves, but he hardly ever gets entirely well. The cases in which recovery is complete are those of simple exudative inflammation, without any necrosis (see Acute Curable Atrophic Paralysis). Much can be done by careful and persistent treatment and by the help of orthopœdic surgery, even in old cases.

The usual course is for the patient to get back the use of all but one leg. He grows to adult life with this short and weakened member.

Treatment.—In the acute stage the child should be put to bed and kept there. Iodine or mustard plasters or leeches must be applied to the spine. Internally, a smart laxative and a diuretic must be given (calomel, gr. iij.; tartrat. potas., gr. xx.). Then tincture of aconite is to be administered in doses of one drop every half-hour as indicated by the fever. To this may be added sweet spirits of nitre. Rest is the most essential thing. The limbs should be kept quiet and warm. At the end of two weeks electrical applications may be very cautiously made to the limbs three times a week, if there is no tenderness or fever. After four weeks electrical treatment should be given daily for a month, each limb being treated for only two or three minutes. After a rest of a fortnight another four-weeks treatment may be given. Treatment should be thus applied intermittently till the end of a year. After this it can be continued or stopped according to the condition of the patient. In old cases daily treatment for one or two years will sometimes produce valuable results (G. M. Hammond). That form of electricity which causes muscular contractions most easily should be employed, and this is usually

the galvanic current. Massage is a most important adjuvant to electricity. It is best given daily for not over ten minutes to a single limb. It is imperative also that the physician overcome any contractures which develop by splints, rubber muscles, and, if necessary, tenotomy. Warmth is very useful. The leg should be bandaged in cotton at night, and, if necessary, hot-water bottles placed beside it. Many parents cannot afford prolonged electrical and massage treatment. In such cases they should be told to rub the limb twice daily with a stimulating liniment and wrap it in cotton or hot flannels at night.

The child should be taught to walk and exercise the limb as much as possible. Tricycles and gymnastic apparatus may often be brought into use here.

Medicines are of little value in the chronic stage. Phosphorus, strychnia, iron, arsenic, cod-liver oil, physostigma, have been recommended and are sometimes prescribed for the improvement of the general health and *solatii causa*.

Chronic Anterior Poliomyelitis.—This form of myelitis is very rare, and most careful examination must be made to exclude on the one hand multiple neuritis, and on the other progressive muscular atrophy. A subacute poliomyelitis has been described, but it really belongs to the acute forms.

Etiology.—Adults are chiefly affected, and men more often than women. Exposure, lead-poisoning, and syphilis are among the principal causes.

Symptoms.—The disease affects one or more of the extremities, often all four of them. There is a gradual paralysis, rapidly followed by atrophy, with degenerative electrical reactions. There is but little pain or other sensory disturbance. The sphincters are not affected.

The disease takes one of two courses:

1. After reaching its height, improvement gradually sets in and recovery may become nearly complete.

2. The disease steadily progresses until the patient presents the picture of a case of progressive muscular atrophy. In a few months or at the most one or two years death ensues. Occasionally, however, after reaching a very advanced stage, the process stops (Fig. 96) and a slight improvement may set in. These progressive cases of chronic poliomyelitis appear to stand half-way between ordinary chronic poliomyelitis and progressive muscular atrophy (see Chronic Atrophic Paralysis of Erb).

Diagnosis.—The disease is distinguished from multiple neuritis by the absence of pain, tenderness, and anæsthesia; from progressive muscular atrophy by the rapid onset, the occurrence of paralysis first and wasting afterward, the early degenerative re-

actions, and the absence of fibrillary contractions. A history of lead-poisoning may also help in the diagnosis.

The *treatment* is mainly symptomatic and must be carried out on the lines indicated under the head of Acute Anterior Polio-

Fig. 96.—Case of Chronic Anterior Poliomyelitis in an Adult, showing the wasted hand and drop foot.

myelitis. Iodide of potassium, mercury, and strychnine should be given.

Minor and Rare Forms of Poliomyelitis.

I. *Acute Curable Atrophic Paralysis* (Landouzy and Dejerine).—The disease comes on rapidly with little constitutional disturbance; it affects profoundly nearly all the muscles of the four ex-

tremities, but does not pick out groups. The cranial nerves are not involved; wasting and degenerative reactions are present. There is no sensory disturbance or involvement of the sphincters. It is a simple acute exudative inflammation, probably of toxic origin, and should be classed with the other cases of anterior poliomyelitis.

II. *Chronic Atrophic Paralysis of Erb.*—This is a disease having a type half way between chronic anterior poliomyelitis and progressive muscular atrophy. The name had best be given to that form of disease which, starting like poliomyelitis, ends as a progressive muscular atrophy.

III. *Subacute Spinal Paralysis of Duchenne.*—The diseases described under this head are chiefly cases of multiple neuritis. In a very few there are both neuritis and myelitis; in others the condition is one of minute focal spinal hemorrhages with secondary myelitis. There is probably no such thing as a "subacute myelitis."

Senile Paraplegia.

This is a form of paraplegia occurring in persons over the age of fifty or sixty. It begins with simple weakness of the legs, followed by wasting and progressive development of a paraplegia. The sphincters become eventually involved. The disease affects the lower extremities first, but gradually extends, and finally involves the arms. The general characters are those of a progressive muscular atrophy; but the disease is distinguished from this by the fact that the sphincters become rather early involved, and that the paralysis and wasting go on together without any fibrillary contractions. The medulla and the facial and ocular muscles do not become involved. Clinically the disease cannot be distinguished from a chronic anterior poliomyelitis which takes upon itself a progressive type. On post mortem, however, it is found that there is a softening of the gray matter in the anterior horns of the spinal cord, more marked in the lumbar swelling. This softening is apparently due to the thickening and obliteration of the blood-vessels from senile changes in them. Dr. Gowers describes a disease which he calls senile paraplegia in which there is simple weakness of the legs and slowness of movement, without any atrophy, sensory disturbance, or alteration in the reflexes. He considers it to be a form of paralysis agitans. The disease which I have described, however, represents more truly a simple senile paraplegia. Very little can be done for this trouble therapeutically. The use of nitro-glycerin, iodide of potassium, sparteine, digitalis, and general tonic and hygienic measures are indicated.

Acute Ascending Paralysis (Landry's Paralysis).

Acute ascending paralysis is a disease characterized by a rapidly developing paralysis which begins in the legs and then involves in turn the trunk, arms, respiratory and throat muscles, usually ending in death. There is little disturbance of sensation, no atrophy or changes in electrical irritability, and no involvement of the sphincters.

Etiology.—The disease is a rare one. It occurs chiefly between the ages of twenty and forty; men are affected oftener than women. Exposure is an exciting cause, and it occurs sometimes after acute infectious fevers and syphilis. The form of rabies known as "paralytic" causes a disease which is apparently identical with Landry's paralysis.

Symptoms.—There may be slight premonitory symptoms for a few days, consisting of numbness in the extremities, pain in the back or limbs, and malaise. The first definite sign of the disease is weakness in the legs, which rapidly increases until in a day or two the patient cannot walk. The paralysis soon involves the arms and then the muscles of respiration; the medulla is last affected, and then respiration becomes difficult, swallowing and articulation may be impossible. In rare cases there are facial and eye palsies. During the course of the paralysis there is little pain or sensory disturbance, but some degree of anæsthesia may occur. The deep reflexes are abolished. There are no vasomotor and no secretory disturbances, no noticeable atrophy, and no degenerative reactions in the affected muscles. The bladder and rectum are involved only in rare cases. There may be slight initial fever, but none occurs after the disease has well set in. The mind remains clear.

The disease, as a rule, ends fatally, and it usually runs its course in less than a week. Death has occurred in forty-eight hours. On the other hand, death has been postponed three or four weeks.

Variations.—The disease has been known to begin in the medulla or cervical region and descend.

Pathological Anatomy.—A number of different diseases have been described under the head of Landry's paralysis, and correspondingly a number of different anatomical changes have been found. Multiple neuritis, acute diffuse myelitis and poliomyelitis existed in some cases. In others there was a dropsical exudation in the central canal of the spinal cord, or a hyaline change in the central arteries. In most instances no anatomical change is present.

It is probable that in true Landry's paralysis there is no lesion

unless the disease is severe and prolonged. The paralysis is due to a poison of microbic origin. In some cases certainly this poison is that of rabies; but it is not impossible that other infections may pick out and suspend the functions of the anterior cornual cells, or, as Gowers suggests, the "end-brushes" of the motor tract which connect with these cells. This would explain the symptoms.

Sometimes the poison may be so great in amount and so irritating as to set up a myelitis or perhaps a neuritis. Cases illustrating these facts have been reported Eichberg, Rosenheim, Putnam. But in most cases the patient dies before the toxin can produce any inflammatory reaction.

The prognosis is very grave, but not absolutely bad. If there is reason to suspect the case of being one of paralytic rabies, no hope can be offered.

Diagnosis.—This must be made from acute poliomyelitis, acute myelitis, acute multiple neuritis.

Its acute ascending course, absence of fever, of anæsthesia, atrophy, decubitus, sphincter troubles, and especially the absence of degenerative electrical reactions are sufficient to enable one to make the diagnosis.

Treatment.—This consists of warm baths or packs, counter-irritation to the spine, laxatives, and rest. Large doses of ergotin, gr. ij., every hour have been successful in one case. Salicylate or benzoate of soda may be tried.

CHAPTER XIII.

THE DEGENERATIVE DISEASES OF THE SPINAL CORD.

Introductory: The Nature and Types of Degeneration and Sclerosis.

THE degenerative diseases of the spinal cord are sometimes called "system diseases," and some are often spoken of as scleroses. There are no true system diseases, however, except locomotor ataxia, progressive muscular atrophy, and amyotrophic lateral sclerosis. These diseases attack certain columns or parts having a definite physiological purpose. Still, even they are not strictly confined to these parts, and the term "system diseases" is best not used as a basis of classification. The term "sclerosis," also, is somewhat misleading. It is not a proper word to indicate the pathological condition, but it has become generally adopted. Sclerosis is the fibroid (and neuroglia) induration which results from degeneration, destruction, or inflammation. We speak of *degenerative* sclerosis, of an *inflammatory* and of a neuroglia sclerosis, or of a sclerosis of mixed origin, according to the nature of the primary disease. The words "degeneration" and "sclerosis" are often used to indicate the same thing, one being the pathological name, the other the anatomical. I shall use the term "sclerosis" here in presenting a classification of the degenerations and scleroses of the spinal cord.

- **Spinal scleroses.**
  - **Primary.**
    - Posterior spinal sclerosis (locomotor ataxia).
    - Combined sclerosis.
    - Multiple sclerosis.
    - Progressive muscular atrophy.
  - **Secondary.**
    - Ascending and descending degenerations.
  - **Inflammatory and mixed.**
    - Chronic myelitis and sclerosis following destruction of cord.

I have already described the scleroses following necrosis and inflammation, the secondary degenerations and scleroses con-

nected with these processes are described elsewhere. Multiple sclerosis is a cerebro-spinal disease and will be discussed in a later chapter. We have therefore now to consider the primary scleroses.

The *primary degenerative scleroses* are due to degenerations which begin in the nerve tissue and end in its atrophy, with substitution of connective tissue. As to their *nature* so far as the microscope shows us, it is a gradual decay and death of the nerve fibre and cell. In some fibroid processes, like locomotor ataxia, this decay is accompanied with the development of irritating products, leucomaines or toxalbumins, which may produce so active a change in the connective tissue as to lead to something resembling a secondary or reactive inflammation. This is never of high grade, however, and in some forms of tabes is very slight.

In progressive muscular atrophy the decay and death produce few irritating products, though enough, perhaps, to account for the fibrillary twitchings and occasional hypertonic condition of the muscles.

The ultimate cause of these degenerative processes is not known. The progressive character of the diseases like locomotor ataxia and progressive muscular atrophy would lead one to think that there is a poison at work and constantly acting on the diseased tissue.

So far, all bacteriological examinations have failed to discover any microbe, but the fact that many degenerative processes follow infectious fevers or syphilis has led to the suggestion that pathogenic germs have poured into the system a poison, or have so modified the cellular nutrition that there is a poison constantly thrown out which irritates and destroys certain areas of nerve tissue.

All the primary degenerations or scleroses have a certain degree of kinship. Their causes are in many respects the same, the course of all is uniformly progressive, and one not very infrequently complicates another. The sharpest distinctions are found between those affecting the gray matter and those affecting the white. Degenerative processes implicating the former tissue are much rarer and their course is more rapid and fatal. It is the anterior and central parts of the gray matter which are almost exclusively affected; consequently we have clinically only progressive muscular atrophy and its allied forms, bulbar paralysis, ophthalmoplegia externa, and amyotrophic lateral sclerosis.

The degenerations of the white matter are more common, slower in course, different in etiology, and much more varied in symptomatology. As a rule, the gray matter is not much involved except secondarily and late.

LOCOMOTOR ATAXIA (Posterior Spinal Sclerosis; Tabes Dorsalis).

Definition.—Locomotor ataxia is a chronic progressive disease of the spinal cord and peripheral nerves, characterized clinically by inco-ordination, pains, anæsthesia, and various visceral, trophic, and other symptoms, and anatomically by a degenerative sclerosis chiefly marked in the posterior columns of the cord and posterior roots, and to a less extent in the peripheral nerves.

Forms.—Besides the common and typical form, there are anomalous and complicated types.

Types.
1. Common form.
2. Neuralgic.
3. Paralytic.
4. With initial optic atrophy.

Complicated forms.
With muscular atrophy.
With other scleroses.
With general paralysis.

Etiology.—The disease occurs oftenest in middle life, between thirty and forty, next between forty and fifty. It may occur as early as the tenth and as late as the sixtieth year. In the very early cases it is usually due to hereditary syphilis. It is much more common in males. Hereditary influence is very unimportant and is only indirect, *i.e.*, the parents may be neurotic. Diathetic influence is slight. Exposures to wet and cold, combined with muscular exertions, are effective causes. Soldiers, travellers, and drivers are rather more susceptible. Excessive railroad travelling, excessive dancing with exposure, favor the development of the disease. Excessive sexual intercourse, combined with irregular living, is a predisposing cause. Syphilis is a very important factor. A history of the disease is obtained in from one-half to two-thirds of the cases. In my cases about forty per cent had had syphilis. The patient usually has contracted the venereal disease ten to fifteen years before, and has rarely had noticeable secondary symptoms.* Syphilis is not a direct factor, but prepares the system for the degenerative process. Syphilis followed later by excesses—mental or physical—and by exposures espe-

* Among 92 personal cases there were 85 males, 7 females. Ages when disease began: Twenty-one to thirty, 15; thirty-one to forty, 38; forty-one to fifty, 29; fifty-one to sixty, 8; sixty-one to seventy, 2. Syphilis in 35; doubtful, 5; denied, 43; unknown, 9. Americans of three generations or more, 15; English, 6; Scotch, 2. The rest about equally divided between Irish and Irish-Americans, German and German Americans. Among Americans the disease apparently begins earlier, and paralytic forms are more common. The duration when seen had averaged five or six years, ranging from one-half year to twenty-five years. The time between infection and the beginning of the disease ranged from one and a half to twenty-four years, averaging thirteen years.

cially tends to produce locomotor ataxia. Lack of proper iodide treatment also favors it.

Among other causes are profoundly depressing emotions, acute infective diseases like typhus, pneumonia, and rheumatism, difficult labors with severe hemorrhage, prolonged lactation, injuries with shock, and excessive smoking.

Locomotor ataxia in a somewhat atypical form may result secondarily from gummatous inflammation of the spinal meninges, from a tumor, and possibly from an ascending neuritis.

Symptoms.—The disease is generally divided into three stages: the initial or præataxic, the ataxic, and the paralytic.

1. The initial stage. The patient first notices a slight uncertainty in walking, especially at night; he has numb feelings in his feet, and at times darting pains in the legs or rectum. His sexual function becomes weak, his control over the bladder slightly impaired. He has temporary attacks of vertigo and of double vision. A continuous sense of profound weariness oppresses him, even though he has made no exertion. The knee jerk is lost. Such symptoms may last a few months or several years.

2. The ataxic stage. The gait now becomes so unsteady that others notice it; the patient has to use a cane, and when walking watch his feet and the ground. If he stands with his eyes closed, he totters and may fall. His feet feel as though there was a layer of cloth or cotton between the soles and the ground. Paroxysms of lightning-like pains attack the legs. Tests show anæsthesia present in the toes and feet or in patches on the legs. A sense of constriction is felt around the waist. The sexual power is often lost; the bladder is weak, and care has to be taken to empty it. The bowels are constipated; at times he has attacks of intense pain in the epigastrium, with vomiting and perhaps a diarrhœa coming on without cause. The pupils are small and do not react to light, but do react to accommodation; vision is still good. The inco-ordination and pain and anæsthesia after a time begin to affect slightly the arms. This stage lasts several years.

3. The paralytic stage. After several years with various remissions and improvements, the patient loses the power of walking altogether. His legs are somewhat wasted, but the muscular strength is fairly good. The anæsthesia and ataxia are very great. The patient does not feel the prick of a pin or touch of the hand; nor with closed eyes does he know where his legs are. His bladder is anæsthetic and paretic, so that the urine has to be drawn. The pains are much less, but are still present at times. The arms are more involved, but never so seriously as to make them useless like the legs. The intelligence remains good, and the patient may continue bed ridden for years, dying finally from some intercurrent affection.

The following table shows the prominent symptoms in the usual order of their appearance:

	First Stage. (Half to twenty years.)	Second Stage. (Two to ten years.)	Third Stage. (Two to ten years.)
Motor	Eye palsies Ataxia Muscular weakness	Less Increased. Paresis	 Increased. Paraplegia.
Sensory	Pains	Pains Anæsthesia	Pain less. Increased.
Excito-reflex	Loss of knee jerk A. R. pupil		
Trophic	Arthropathies	More rare	Rare.
Visceral	Sexual weakness Vesical weakness Constipation	Increased	Increased.
Special senses	Diplopia Optic atrophy	Rare Rare Deafness	 Increased. Increased. Paralysis of accommodation.

The symptoms must now be analyzed more closely.

Locomotor and *static ataxia* are present very early, but only to a moderate extent. Tests such as making the patient walk and stand with the eyes closed, noting the position of limbs and the weight of objects, will reveal an ataxia due largely to begin-

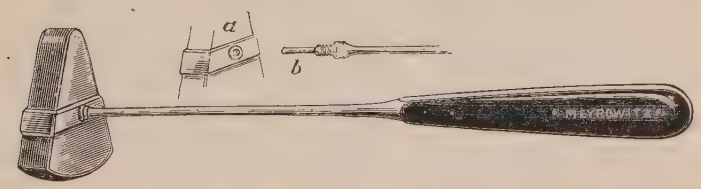


Fig. 97.—Instrument for Testing Tendon Reflexes.

ning anæsthesia of the joints and tendons. By the use of the ataxigraph, one can with care assure himself that the patient has an excessive degree of static ataxia. In my experience, when the ataxigraph records over three inches' oscillation, the patient not being paraplegic or under the influence of any drug, it is abnormal.

The patella-tendon reflex or knee jerk is abolished very early in all typical cases. This constitutes a very important symptom, therefore.

The gait and station in ataxia are characteristic. In walking, the patient keeps his eyes on the ground and on his feet. The latter he throws out rather forcibly, owing to overaction of the extensors of the foot. In watching such a patient walk bare-

footed, the extensor tendons can be seen to stand out with each forward movement of the limb. The foot is brought down sharply on the heel and the legs are spread apart a little. Turning a corner, turning around, and going downstairs are done awkwardly, and the patient is apt to totter and fall. Walking on a chalked line is very difficult; so also is walking backward. The gait improves after the patient walks awhile, and he will generally say that the practice of walking does him good. Still, he soon gets tired (Fig. 98).

Fig. 99.—Showing Station in Second Stage of Locomotor Ataxia.

Severe rectal *neuralgia*, associated perhaps with hæmorrhoids, is sometimes an early symptom. Persistent neuralgia and functional disturbance of the bladder and rectum should cause suspicion of ataxia. Lancinating or lightning pains occur and are very characteristic. The pains dart down the legs along the course of the sciatic, or they suddenly appear as patches of pain on the foot or leg or thigh ("spot pains"). The pain comes unexpectedly and with such severity that the patient involuntarily jumps or jerks the limb. He speaks of his "jerking" and "twitching pains." The pains may affect the bowels or be felt as a squeezing sensation around the waist (girdle pains). The pains of ataxia are often the most obstinate and distressing symptom. They usually come on in great intensity once or twice a month, and last for two or three days. They then leave the patient for a time. They are often worse in cold and damp weather. In some cases the pains are almost continuous, coming on, if not every day, at least two or three times a week. Such cases are associated with much cutaneous hyperæsthesia, especially during the attacks. This type of cases is called "*the neuralgic*." The patients rarely have as much ataxia, paresis, or visceral troubles as do the typical forms, and in certain respects such cases are favorable.

The pains of the disease continue well into the second and even

third stage. Meanwhile the anæsthesia becomes much more marked. It affects most the feet and next the legs, rarely extending much over the thighs, but passing to the fingers and hands. The anæsthesia is greatest to pain, but touch and temperature sense are also involved. There is often delayed conduction and polyæsthesia; many other curious perversions of the cutaneous sense are noted. Some anæsthesia usually develops

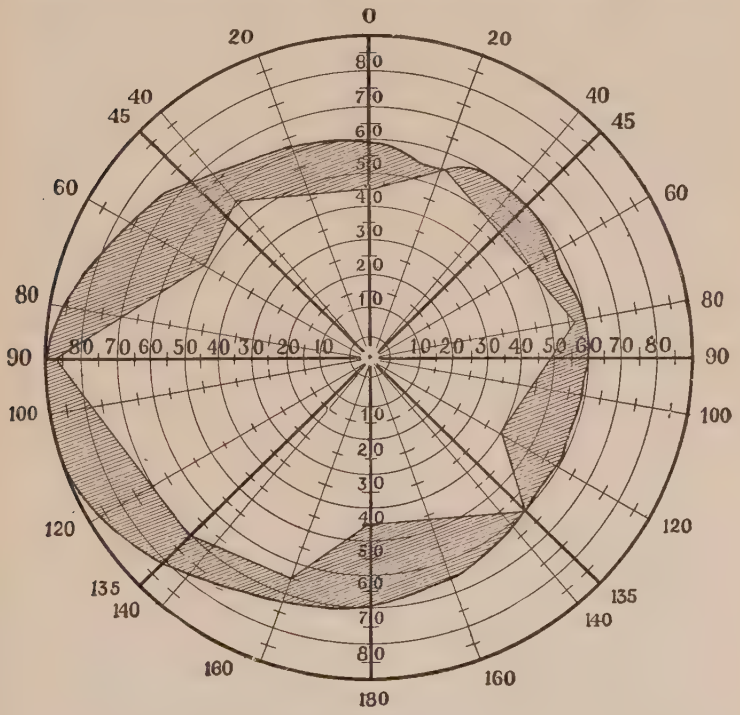


Fig. 99.—Irregularly, Contracted Visual Field in Case of Tabes with Optic Atrophy, Left Eye (Berger).

over the finger tips and hands, and sometimes a band of anæsthesia develops about the waist. The facial and cranial nerves are not much affected, but there may be trigeminal neuralgia.

Optic atrophy occurs in from ten to thirty-five per cent of cases, according to statistics. In my experience the percentage was about ten, the same as that of Gowers. Optic atrophy usually develops in the præataxic stage. If a patient has reached the *second stage without it, he will probably escape it altogether.* Cases with ocular paralyses are slightly more disposed to it

(Berger). It attacks the left eye oftener than the right. The atrophy begins sometimes with increased sensibility to light, flashes of light, and muscæ volitantes. With the failing vision, disturbance of color sense often and contraction of the visual field always occur. This contraction is irregular, with sector-formed defects; not hemiopic (Fig. 99). The atrophy progresses slowly with slight remissions. It may cease its progress, but this is rare. Blindness comes in about three years. Ophthalmoscopically there may be seen slight evidence of congestion in the early stage; later, pallor of the disc, which finally becomes grayish.

Disorders of *hearing* are frequent in tabes, occurring in about one-fourth of the cases, but in the majority of instances the aural trouble is an accidental complication due to middle-ear disease. Primary atrophy of the auditory nerve is very rare, as might be expected, since this nerve is structurally not like the optic nerve. Its existence has been inferred on clinical grounds. Another form of tabetic deafness is of trophic origin and due to a sclerotic condition of the middle ear (Treitel). It is caused by involvement of the trophic or vasomotor fibres of the fifth nerve.

The senses of *taste* and *smell* are rarely affected.

The *eye muscles* are implicated in some way in nearly all cases of tabes. The following are the disorders:

1. Loss of the light-reflex, and myosis. 2. Sympathetic-nerve ptosis. 3. Paralysis of branches of the third nerve. 4. Paralysis of the sixth nerve.

Paralyses of the ocular muscles (third and sixth) occur rather oftener in syphilitic cases. Other ocular troubles are not influenced by a preceding syphilis. Ocular palsies occur relatively about equally often at the various ages at which tabes develops. They are early symptoms of the disease occurring as a rule in the præataxic stage.

1. Loss of light-reflex and pupillary rigidity. The pupils are small and sometimes uneven; they do not respond to light, but they do to accommodation. This condition is known as the *Argyll-Robertson* pupil. In early stages the light-reflex may be simply sluggish. In the late stages the accommodation-reflex is also lost. The Argyll-Robertson pupil is practically only found in tabes and in general paresis. The ocular skin-reflex usually disappears early. The myosis in tabes is due to paralysis of the sympathetic dilating fibres. The pupils are sometimes irregular in shape.

2. Sympathetic-nerve ptosis. A slight drooping of one or both lids is not infrequent. It begins early and progresses slightly up to the later stages of the disease. It is due to paralysis of the sympathetic-nerve fibres of the lid.

3, 4. Paralysis of the external eye muscles. The external rec-

tus is oftenest affected of single muscles, but the various branches of the third nerve taken together are oftener involved than the sixth. Of the third nerve's branches, the levator palpebræ and internal recti muscles are oftenest involved. There may be multiple palsies. These occur oftener in syphilitic cases. Progressive ophthalmoplegia may be associated with tabes. The ocular nerve palsies may be transitory or permanent. Those occurring in the præataxic stage are usually transitory, taking a few hours, days, or weeks. Cases have even lasted two years and got well. The permanent palsies develop usually in the later stages.

The *arthropathies of locomotor ataxia.* Degenerative diseases of the joints, technically known as arthropathies, and spontaneous fractures of bones form important symptoms of tabes. They occur in ten per cent (Charcot) or five per cent (author) of cases.

The arthropathies are three or four times more frequent than the fractures. The joints oftenest affected are the knees, ankles, and hips; but the elbow, shoulder, wrist, and small joints may be attacked.

Spontaneous fractures occur oftenest in the shaft and neck of the femur, next in the legs, forearm, humerus, and clavicle. The pelvis, scapula, vertebræ, and under jaw may be fractured. Arthropathies are often accompanied with fractures, especially of the heads of the bones. The two sides of the body are about equally affected.

Fig. 100. — Arthropathy of Ankle.

Fig. 101.—Arthropathy of Knee (A. S. Roberts).

The arthropathies are characterized by a sudden, apparently spontaneous painless swelling of the joint. The symptoms may develop in twenty-four or forty-eight hours. In rare cases there is a history of some preceding rheumatic pains or of an injury. After a time there is an osseous hyperplasia of the joint, which becomes enlarged to enormous proportions (Fig. 101). There is also a tendency to luxation of the joint. It crepitates on moving. There is no tenderness on pressure, the hand finds evidence of synovial exudation, roughened surfaces, and perhaps

fractures of the enlarged parts. In the milder forms there is simply swelling from synovial exudation and some enlargement of the bones with roughened surfaces. After a few weeks this swelling may subside and the joint return to nearly its natural size. In other cases the process progresses, the ligaments relax, the bones of the joint can be moved about freely, and luxations are easily produced. There is still no pain, but the limb becomes

FIG. 102.—ARTHROPATHY INVOLVING KNEES AND LONG BONES OF LEGS.

almost or entirely useless on account of the loose and relaxed condition of the parts (Figs. 101, 103).

As time goes on, some absorption takes place and the head of the bone may almost disappear. The arthropathies have been divided into benign and malignant, but no sharp line can be drawn or certain prognosis made in the early stage.

The arthropathies appear in the prodromal and early stage of the disease in over half the cases, and are often at first unrecognized. One-third occur after the tenth year of the disease.

The spontaneous fractures are usually brought on by a slight trauma, such as a fall. Violent muscular movements may produce them. They also are painless as a rule. The fractures usually heal well, often with abnormal readiness, but occasionally there is delay, and often healing is accompanied with great throwing out of callus.

Pathologically the arthropathy is a rarefying osteitis. It does not differ anatomically from arthritis deformans, except that fractures may accompany it. Clinically the chief difference lies in the abruptness, spontaneity, and painlessness of the process. The disease, on the whole, cannot be considered specifically different from arthritis deformans, modified by the analgesia of the parts. It is due probably to a degenerative change in the nerves supplying the joints and bones. The process may begin in the cartilage, bone, or ligaments. Eventually all these parts are involved. There is congestion of the synovial membranes with hydrarthrosis, then atrophy and rarefying hypertrophy of the epiphyses, relaxation of the ligaments, formation of osteophytes and bony-stalactites. There may be a rarefying osteitis of the long bones, without much joint involvement at first (Fig. 102).

FIG. 103.—ARTHROPATHY OF ELBOW (A. S. ROBERTS).

Various *trophic disturbances* of the skin may appear, generally late in the disease. The most common are herpes and lichen. Besides these, bullæ, transitory erythema, urticaria, eczema, pemphigus, ecthyma, ulcers, ichthyosis, and petechiæ have been described; but they are rare and often only accidental complications. A peculiar round, perforating ulcer sometimes develops on the sole of the foot, often as the result of cutting a corn. In rare cases the nails and teeth fall out. In syphilitic cases there is usually baldness.

Peculiar "crises" of various kinds occur in tabes. The most common are *gastric crises*. These consist of attacks of intense pain extending from the groin to the epigastrium or encircling the waist, accompanied with vomiting and sometimes diar-

rhœa. The attacks are usually associated with pains in the legs. They last two or three days, then pass away.

Laryngeal crises consist of attacks of spasm of the adductors or paralysis of abductors, with noisy, croupy respiration. The attacks come on suddenly, the patient coughs and struggles for breath, and he may be seized with vertigo and fall down. The pulse may be very fast. The paroxysm lasts for a few minutes to several hours. The symptoms are very distressing, but not dangerous. Paroxysms of cough have been described as "bronchial crises." There are also *cardiac crises*, in which there is dyspnœa and rapid heart-beat and sense of suffocation resembling angina. The heart itself sometimes is diseased, but whether from neurotrophic disturbance or not is doubtful. The pulse is often small, rather rapid, and weak.

The laryngeal and heart crises both depend on a degenerative irritation of the vagus, and may be more or less united in symptoms.

A *sense of great weariness* and heaviness in the limbs, present constantly, no matter how much rest is taken, is a characteristic symptom, and is due to an irritability of the nerves of muscular sensibility.

Muscular atrophies occur sometimes in tabes. They are of three kinds: 1st, a true progressive muscular atrophy due to degeneration of trophic and motor cells; 2d, localized muscular atrophies due to degenerative atrophy of nerves; 3d, a general wasting.

Under the first head one finds ophthalmoplegia, bulbar paralysis, and spinal amyotrophy. Under the second, wasting of certain groups of muscles in the legs or arms.

Besides these, there is a generalized atrophy which occurs in the paralytic stage and is due probably to a slight involvement of the anterior horns in the progressive process that affects the cord.

Attacks of hemiplegia in rare instances occur in tabes. They are usually of temporary character and occur early in the disease. They may come on late and are then more likely due to acute softening.

Acute paraplegia comes on occasionally also, and this sometimes almost disappears.

The *sexual power* may be at first greatly exaggerated; but this is rare, and usually there is progressive weakness and loss of desire. The bladder and sexual functions are rarely entirely lost and rarely equally impaired in this stage; one may continue good while the other is affected moderately. Usually the sexual function goes first.

Some *cerebral symptoms* occur in tabes, chiefly in the early stage. They are insomnia, which may be very obstinate, and occa-

sional vertigo. An irritability of temper and tendency to despondency, sometimes noted, cannot be considered unnatural. Apoplectiform and epileptiform attacks are described, but are very rare, and should cause a suspicion of a complication. The disease in very rare cases terminates in general paresis.

Course. — The disease has been termed progressive, but it is not so in a large number of cases. With proper treatment the symptoms can often be kept in control for years. The first stage may last twenty years or more; the second stage five to fifteen years. The total duration of the disease varies enormously, ranging between three and thirty years. A few acute cases have been observed, running a course of less than a year.

Complications. — These are acute myelitis, generally syphilitic; lateral sclerosis, progressive muscular atrophy, hemiplegia from embolism or endarteritis, general paresis, and heart disease.

Pathological Anatomy and Pathology.—The characteristic changes are found in the spinal cord and posterior roots, and to a less extent in the peripheral nerves. The spinal cord usually is reduced in size and flattened antero-posteriorly;

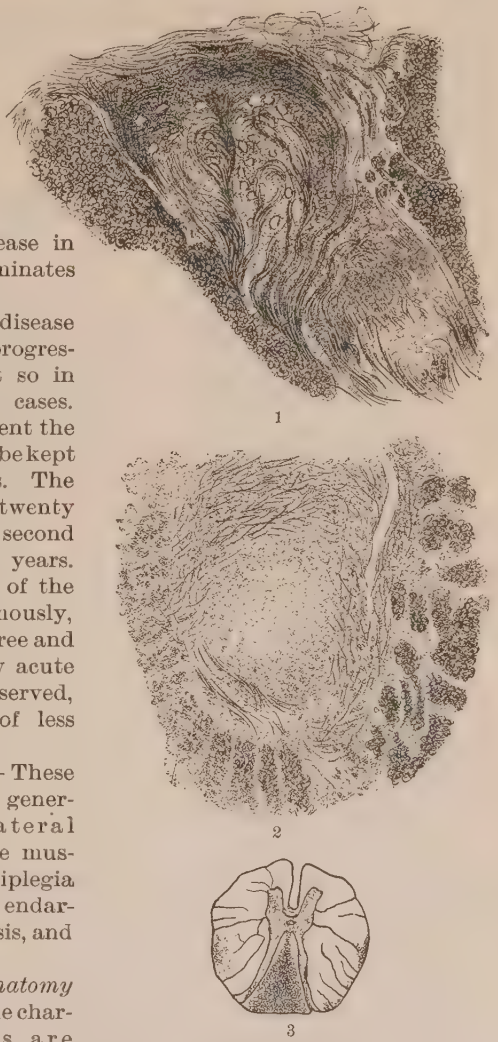


Fig. 104.—Showing Columns of Clark. 1, Normal; 2, diseased, in case of tabes in third stage; with degeneration of posterior columns and cerebellar tracts as shown in 3 (Oppenheim).

the pia mater is thickened somewhat. One can see with the naked eye that the posterior columns of the cord are shrunken and have a grayish appearance.

Under the microscope it can be seen that the white matter of the posterior columns is very seriously affected; the nerve tissue has disappeared, and its place is taken by connective tissue through which a few nerve fibres still run. The walls of the

Fig. 105.

Fig. 106.

Fig. 105.—Posterior Spinal Ganglion in Third Stage of Tabes. P. R., Posterior root; A. R., anterior root (Oppenheim).

Fig. 106.—Healthy Spinal Ganglion.

blood-vessels are somewhat thickened, but not remarkably so, nor is there any notable evidence of congestion or excessive vascular irritation.

The part of the posterior column first affected is a vertical streak lying in the middle root zone beween the posterior median (columns of Goll) and posterior external columns (columns of Burdach) (Fig. 107). The segments first and most affected are those of the upper lumbar and lower dorsal region. Besides this area the rim zone or column of Lissauer is also early involved. As the disease

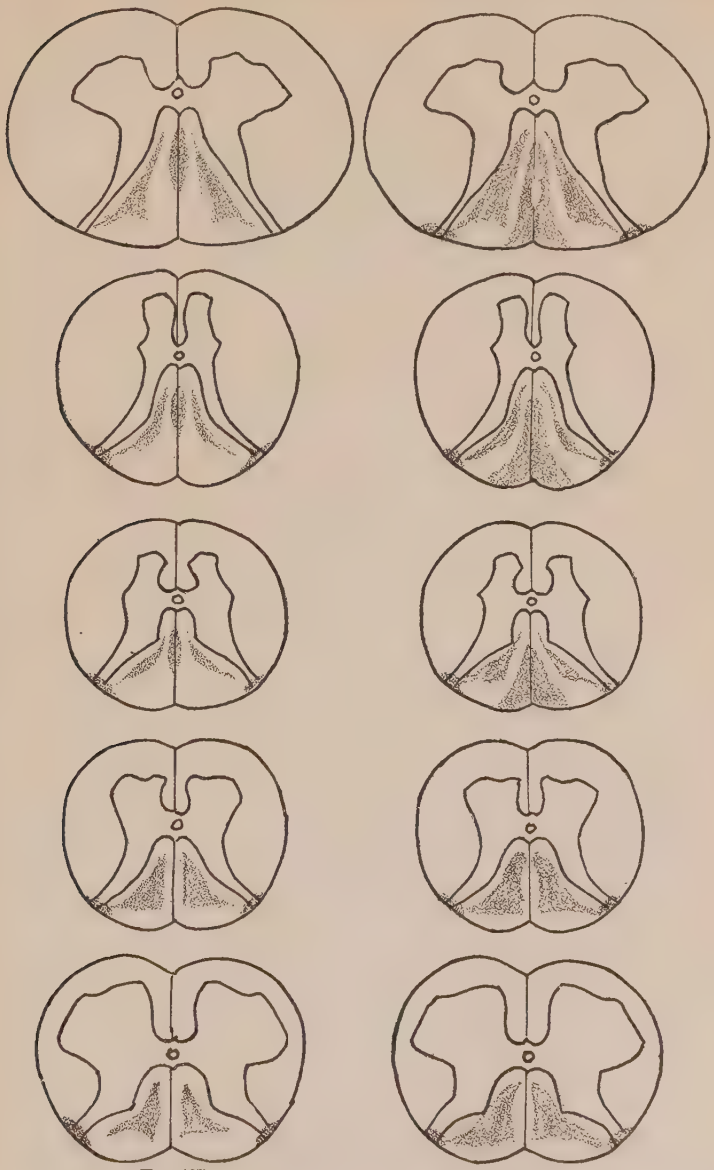


FIG. 107. FIG. 108.

FIG. 107.—LOCOMOTOR ATAXIA, showing areas affected in first stage at five different levels. Drawn from specimens in author's possession and from comparative study of over thirty other figures.

FIG. 108.—LOCOMOTOR ATAXIA, SECOND STAGE.

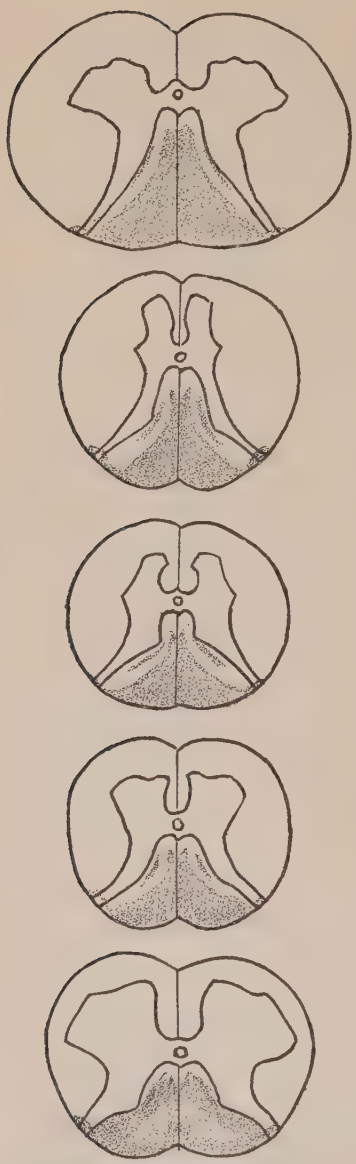


FIG. 109.—LOCOMOTOR ATAXIA, LAST STAGE.

progresses it extends upward and spreads laterally so that finally all of the posterior column is changed into a dense connective-tissue mass through which only a few nerve fibres run. The part last and least involved is that lying just posterior to the commissure (anterior root zone of Flechsig) and that lying just mesial of the posterior horns (external part of the middle root zone). There is sometimes a degeneration of the antero-lateral ascending tract (Gowers' tract) and very rarely of the cerebellar tract. The pyramidal tracts are only involved in complicated cases. The cells and fibres of the column of Clark are often involved in advanced cases (Fig. 104). The gray matter of both the posterior and anterior horns may show some degenerative changes, viz., decrease of the fibre network and atrophy of the cellular elements.

The posterior roots are usually involved, the process extending as far as the spinal ganglia, which also show some degeneration (Figs. 105, 106). The anterior roots are normal.

The process begins in the upper lumbar cord; the sacral cord is usually much less affected. However, the exact initial point of attack varies, and this accounts for the variation in the symptoms. Cases that begin with decided bladder and genital symptoms probably start low down; cases which go for a good while with only ataxia, loss of knee jerk, and

pains begin higher; while in the brachial or arm-type cases the process begins in the cervical enlargement.

The peripheral nerves are diseased in a large number of the

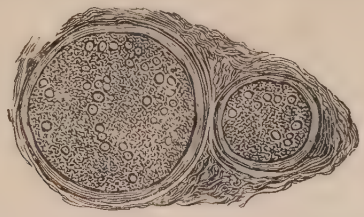


Fig. 110.—Plantar Nerve Simple Atrophy in Tabes.

advanced cases. The nerves of the leg are most involved. The process is a degenerative atrophy or neuritis (Figs. 110, 111). It affects the extremities of the nerves first and slowly extends upward, seldom reaching the large trunks.

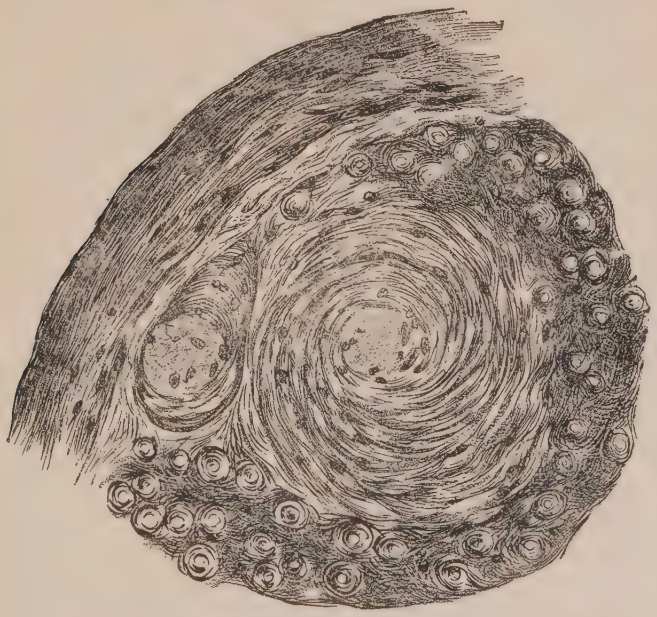


Fig. 111.—Ulnar Nerve Third Stage Tabes, Atrophy with Proliferation of Connective Tissue (Oppenheim).

In the early stage the only nerve that is often affected is the optic. The process here is an atrophy beginning at the periphery and extending brainward. The third, fifth, and sixth nerves are

occasionally involved; still more rarely the olfactory and auditory. The vagus nerve and sometimes its nucleus and that of the glosso-pharyngeal are implicated, it may be, rather early in the disease. It is believed that these facts explain many of the laryngeal and visceral crises.

The disease is primarily an irritative degeneration. The nerve fibres of the cord are first involved. Their destruction is not a simple wasting, but is accompanied with evidence of irritation such as swelling of axis cylinders and, secondarily, proliferation of connective tissue and slight congestion. The disease is spoken of by some as a parenchymatous inflammation (Charcot); by others as a vascular sclerosis; by others as a gliosis, *i.e.*, a substitution of neuroglia tissue for nerve fibre. In the light of present knowledge, however, it is safest to say that, through some toxic or diathetic influence constantly acting, there is set up a progressive destructive process which has a selective influence on certain tracts in the posterior columns with their roots and ganglia and to a less extent on the peripheral nerves, particularly the optic.

The *diagnosis* is not difficult in the advanced stages. In the first stage the disease has to be distinguished from multiple neuritis, chronic myelitis, spinal tumor, spinal syphilis, general paresis, and neurasthenia. Multiple neuritis, in its sensory or pseudotabetic form, sometimes resembles closely locomotor ataxia. The differential points are given under that disease.

In myelitis there is more paralysis, generally exaggeration of reflexes, and an absence of disturbance of special senses.

The diagnostic criteria of locomotor ataxia in all cases are the presence of lightning pains, numbness of the feet, loss of knee jerk, ataxia of station and gait, without much loss of muscular power, the presence of the Argyll-Robertson pupil, the history of syphilis, and the slow onset of the disease.

Prognosis.—In the first stage a small percentage may have the disease stopped and get practically well. After the second stage a cure is impossible, but great improvement may be secured and the patient made relatively comfortable for years.

In the third stage little can be done except relieve the symptoms, but life may be prolonged. Death usually occurs from some intercurrent malady, or from kidney disease caused by the bladder trouble. Patients very rarely indeed die from the disease itself and its various "crises."

Treatment.—The hygienic treatment consists in enforcing a very regular and quiet life. The meals, sleep, work, and exercise should be taken in the most systematic way. It is often best when possible to have the patient give up business. This is especially necessary if the business involves great mental and phy-

sical strain. Absolute rest in bed for several months is often very helpful, especially in neuralgic cases. Out-door country life is better than city life. Climates of low altitude, if not very moist, are better than high altitudes. Sea voyages are often helpful.

A diet which is non-fermentative and easily digestible, *i.e.*, a nitrogenous diet, is preferable. The absolute diet of meat with hot water seems rarely indicated, and has not proved useful in my experience.

Hydrotherapy is much employed in Germany. I have found lukewarm baths at a temperature of 95° F., for ten or twenty minutes daily, helpful in some cases, especially in early stages. Later, cold affusions over the back may do much good.

Electricity is of use from its general tonic and reflex effects, and perhaps exercises some direct influence on the diseased process. Strong galvanic currents (15 to 30 ma.) should be applied along the spine, through the trunk, and down the legs and arms. The combined galvanic and faradic current is even better, given in the same way. The faradic brush should be applied over the extremities and along the back.

The actual cautery is efficient in stopping pains. It should be applied to the back as often as once a month at least and sometimes once a week. Dry cups may be applied rapidly and in great number (80 to 100) along the spine and along the course of the sciatic nerves. In very painful cases occasional wet cups and leeches are useful. Blisters and various forms of counter-irritants sometimes do good.

Suspension by the neck and arms is helpful, beyond any doubt, in about fifteen per cent of the cases. It is best adapted to persons in the second stage and to those who have a good deal of bladder trouble and pain. It is of little value in the paralytic stage and must be used with care in the early stage. Suspensions should be given for one to three minutes three times a week until twenty-five or thirty are taken. After three months a second course may be given. The various ingenious modifications of suspension, by which the back is flexed, extended, or stretched by weights and pulleys, have not been shown to have any advantage.

Medicinally, for the diseased condition itself it is customary to give iodide of potash, arsenic, strychnine, phosphorus, nitrate of silver, chloride of gold and sodium, ergot, mercury, and chloride of aluminium. I doubt if any of these drugs are of value except silver, and sometimes the iodide, bichloride of mercury, and gold. Of them silver is the best, and appears undoubtedly to affect favorably the course of the disease. Various salts of silver are used, but none have any special demonstrated value over the

nitrate, which should be given in doses of gr. $\frac{1}{4}$ to $\frac{2}{3}$ ter in die for not over two months at a time. Arsenic is recommended by Gowers, but I have not seen it do any good. The same authority recommends chloride of aluminium in doses of gr. ij. to iv. Symptomatically the drugs most called for are those which relieve pain. Phenacetin and antipyrine stand at the head of the list, but antifebrin, extract of Cannabis indica, codeine, aconitine, exalgin, salicylate of sodium, muriate of ammonia, all may have to be tried. Relief from pain is gotten by using lukewarm baths with pine-needle oil or extract, by applying ice-bags, and by wrapping the limbs in flannel bandages into which sulphur and menthol have been rubbed. Bladder troubles are often much relieved by infusion of buchu ʒ ij. and tincture of hyoscyamus ♏ x. every three hours, or by sandalwood oil. In vesical and rectal neuralgias, suppositories containing iodoform gr. iij., extract belladonna gr. $\frac{1}{3}$, and codeine or antipyrine may be used.

Locomotor Ataxia in Children.

The spinal cord of persons under the age of puberty is not subject to primary systemic degenerative changes. This is a general law to which, aside from the cases of Friedreich's ataxia, there are hardly any exceptions.

The posterior columns alone are sometimes primarily affected; but in almost all instances the disease takes the type known as Friedreich's ataxia, and it may be seriously doubted whether tabes dorsalis ever shows itself in young children in any other way than as hereditary syphilis or as the peculiar form known as degenerative or Friedreich's ataxia.

Still, a few cases have been reported in which an apparently typical tabes began between the ages of nine and twenty.

THE COMBINED SCLEROSES.

By the combined scleroses is meant those forms of degenerative sclerosis in which both the posterior and lateral columns are involved. There are several diseases in which combined sclerosis exists. They are:

1. Ataxic paraplegia
2. Friedreich's ataxia.
3. Complicating scleroses of general paresis.

Besides, there are numerous irregular forms. One of these has a tolerably definite clinical course and may be termed

4. Combined sclerosis with terminal softening. It has been described most fully by J. J. Putnam.

Ataxic Paraplegia (Progressive Spastic Ataxia).

This is a disease characterized by the symptoms of spastic paraplegia and ataxia.

Etiology.—Ataxic paraplegia is not a special and independent disease. It has not a definite pathological basis. It is a clinical symptom-complex to which a special name is given for the sake of convenience. Nearly all cases of ataxic paraplegia are cases of chronic myelitis of the dorsal region, with secondary degenerations, or cases of locomotor ataxia with a complicating or secondary degeneration in the lateral column. In my experience, ataxic paraplegia is oftenest an atypical form of locomotor ataxia. In some cases a myelitic focus may set up true primary degenerative changes in the cord in parts remote from the focus. Bearing the above facts in mind, it is unnecessary to go further into the etiology of the disease, it being caused by the same factors that cause chronic myelitis and locomotor ataxia. It is a disease of early and middle life and of men.

The *symptoms* are those of a gradually developing spastic paraplegia plus more or less ataxia. The first symptoms are a heaviness, numbness, and stiffness in the lower limbs. Ataxia develops early.

It is shown in standing and walking and in attempting to make co-ordinate movements. It is present even when the muscular sense, as tested by posturing the limbs and by weights, is shown to be good. It involves the arms but little. The gait is a combination of that in tabes and in spastic paraplegia. The heels are brought down first in some cases, in others the toe drags and the heel strikes the floor last. Sometimes the whole foot is planted down at once, and the shoes are worn almost evenly.

The deep reflexes are exaggerated and there is trepidation and clonus. Muscular weakness is not very great, but some is present and eventually becomes marked. There is little or no atrophy. There is but little anæsthesia and rarely much pain, nor are there often visceral crises. The eyes are rarely involved. There is early loss of sexual power and often considerable bladder trouble. The disease after a time may involve the arms.

The course is very slow. Some cases eventually lose the spastic symptoms and knee jerks and become typical cases of locomotor ataxia. In other cases the paraplegia increases, contractures set in, and the patient resembles a case in the last stage of myelitis.

Diagnosis.—The clinical diagnosis is easy and unmistakable. The main question is to determine whether one is dealing with atypical tabes or some form of chronic transverse myelitis or

from a focus of multiple sclerosis. In myelitis the symptoms come on more rapidly, the paraplegic symptoms are more pronounced. In some cases, however, a very good clinical picture of ataxic paraplegia follows an attack of acute myelitis. There are very few arm and no cranial nerve symptoms. In the tabetic cases the onset is slower, there is more ataxia and sensory disturbance, and some eye symptoms are usually present.

Pathological Anatomy.—As already stated, the evidence at present shows that ataxic paraplegia of the clinical type described is caused by an extension of the sclerosis of tabes or by a chronic

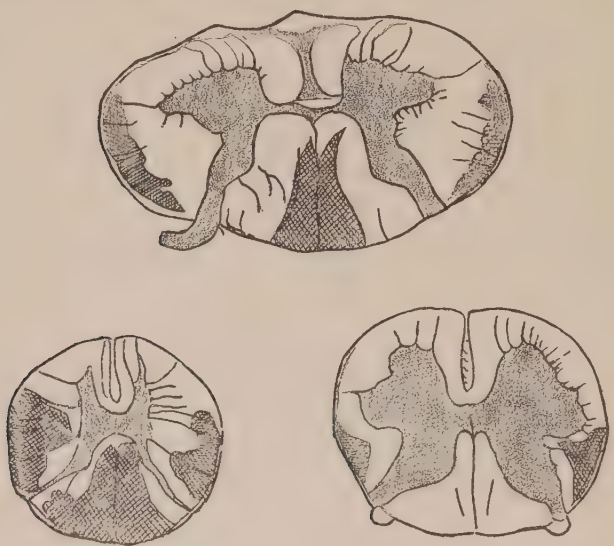


FIG. 112.—DISTRIBUTION OF THE LESION IN ATAXIC PARAPLEGIA OF MYELITIC ORIGIN.

dorsal myelitis. Other conditions causing transverse pressure and destruction of the cord may cause similar symptoms, but they are rare (Figs. 112, 113).

The *treatment* of this trouble does not vary greatly from that of locomotor ataxia or myelitis, and naturally a very important thing is rest. Under this treatment I have seen remarkable amelioration in every symptom. The slight anæsthesia which exists can be made to disappear by the use of electricity. The trembling and trepidation of the limbs at night are often very annoying to the patient. Moderate doses of bromide relieve this.

I can strongly recommend the use of orthopædic appliances for giving support to the shaking knees and hips. Some patients

feel better and are even improved by the systematic, careful use of the faradic current, one of high tension or the static current being preferred. No class of patients, in my experience, has returned so persistently and faithfully for electrical treatment as those suffering with spastic ataxia or spastic paralysis. A thorough course of anti-syphilitic treatment, using inunctions and occasional purges, should be tried if needed, and in later stages very large doses of the iodides are to be tried. In one case, undoubtedly syphilitic, this certainly did much good. The patient preferred iodide of ammonia.

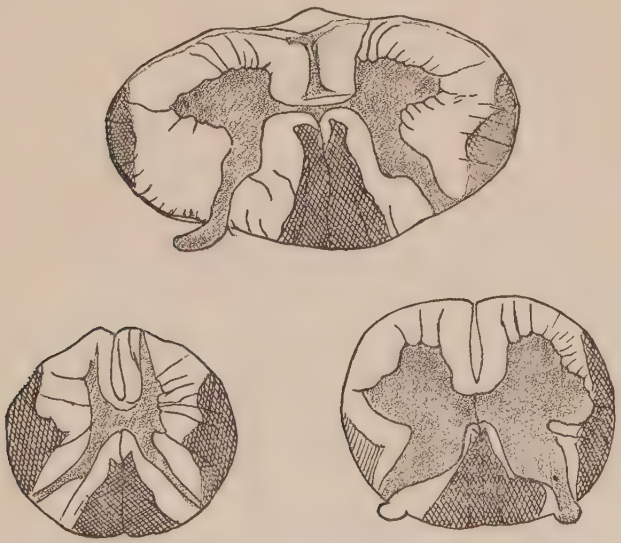


Fig. 113.—Distribution of the Lesion in a Case of Tabetic Origin.

Warm baths and cold douches are beneficial, but hot baths are dangerous. The stomach is a thing that must be attended to with great care.

Friedreich's Ataxia (Hereditary Ataxia, Friedreich's Disease, Family Ataxia, Generic Ataxia).

Friedreich's ataxia is a chronic degenerative disease, affecting the several long-fibre systems of the spinal cord, especially that of the posterior column, and later those of the lateral columns. It begins in the lumbar region and extends upward and downward, finally involving the medulla and especially the nuclei of the hypoglossal nerve. It develops in child-

hood, affecting persons with an imperfectly developed spinal cord the result of a neurotic inheritance. Clinically the disease is characterized by ataxia beginning in the lower limbs and gradually involving the upper limbs and the organs of speech. Curvature of the spine, talipes, vertigo, and finally paralysis and contractures appear. The knee jerk is, as a rule, absent. There is but little pain or anæsthesia, and optic atrophy and visceral troubles are usually absent. The disease is rare, only about two hundred cases being on record.

Etiology.—The fundamental factor in predisposition is an inherited or connate lack of development of the spinal cord, more particularly of the columns of Goll and pyramidal tracts. This condition is inherited directly sometimes, but indirectly as a rule. That is to say, the parents or other members of the family usually show simply a neurotic history, and it is in only a minority of cases that there is a history of ataxia in the direct line of ancestry.

The more frequent condition is this: the patients or grandparents have some neuroses, such as insanity, inebriety, or great nervous irritability; the ataxia occurring only in the children of one generation. Sometimes in a single family the uncles and nephews or cousins may be found to have the disease. Hence the name "family ataxia," used by some writers. There are a good many cases in which the parents were apparently perfectly sound and healthy. Yet it is most probable that the sufferers from Friedreich's disease inherit a tendency to degenerative processes from some of their ancestors. This degenerative tendency may have been shown in those ancestors in a very slight degree. The patients rarely have locomotor ataxia, though this has been observed in a few cases. The children of locomotor ataxics do not have Friedreich's ataxia except in the very rarest instances.

Syphilis in the parents is an element in some—perhaps in many—cases. Habitual intemperance in parents undoubtedly is a factor sometimes; much more rarely consanguinity and tuberculosis act as predisposing causes of degeneration.

More cases have been observed in America than in any other country; while the fewest have been reported from France. The disease develops at about the time of puberty, most cases occurring between the ages of six and fifteen years. It is not very rare, however, for symptoms to develop even in infancy, though some of the cases reported at this time were probably of a syphilitic character. In a given family the disease, as a rule, strikes the older members first, but the younger members are attacked at a relatively earlier age. The most typical time of development is a rather late one, *i.e.*, after twelve years of age. The disease may come on after maturity. In American cases the age of de-

velopment of the disease has been rather earlier than the average. The male sex slightly predominates, its proportion being about sixty per cent. In America the female sex has, however, been more affected (3 to 2). The patients are the children of the laboring and agricultural classes. They have been found in the country oftener than in crowded cities. The families have often been large, but this is not always the case, especially in American cases. Nursing at the mother's breast is thought to have been an exciting cause. Usually the disease appears after infectious fevers such as diphtheria, variola, and typhoid.

Symptoms.—The patient first notices an uncertainty in the gait and some feebleness in the lower limbs. These symptoms gradually increase until they interfere seriously with progression, and force him to leave off active work. With this there may be some slight pains or numbness in the lower limbs, and an examination will show, within a year or earlier, that the knee jerk is gone. After five or six years the arms become affected with incoordination, and a little later bulbar symptoms, such as thick or scanning speech, and often nystagmus, appear. During this time the patient suffers little pain and has no trouble with the bladder or rectum. Vertigo and headache are often present (Fig. 114). Dorsal flexion of the toes, talipes varus or some other form of club-foot, and lateral curvature of the spine are often observed. Oscillation of the head and choreiform or inco-ordinate movements of the extremities may develop. As the disease progresses the legs become weaker, and finally paraplegia, with contractures and muscular wasting, sets in. The disease makes slow progress; often it remains almost at a stand-still for years, and the patients usually die of some intercurrent disease, such as phthisis or an infectious fever.

Among the rarely observed symptoms are tremor, spasms, decreased electrical irritability, muscular atrophy, vasomotor paresis, polyuria, glycosuria, anæsthesia, fibrillary tremor, choking attacks, ptyalism, strabismus, diplopia, blepharospasm, a slight degree of ptosis, sluggish pupils, tachycardia, profuse sweats, impotence, slight vesical incontinence, fragilitas ossium. Many of these symptoms are, however, exceptional and accidental.

The major and essential symptoms are (1) ataxia, beginning in the lower limbs and extending to the arms and tongue; (2) peculiar rolling, ataxic gait, ataxia gradually involving the arms; (3) disturbances of speech; (4) talipes and spinal curvatures; (5) gradual development of paraplegia; (6) loss of knee jerk; (7) absence of cutaneous anæsthesia, of bladder troubles, of eye troubles except nystagmus, and of severe pains; (8) the development of the foregoing at about the time of puberty.

Pathology.—Sixteen autopsies have been made upon cases of

Friedreich's ataxia. The disease in most of the cases had lasted over ten years; in one, only two years; in two, eight years. The examinations have led to quite uniform results. The lesions of importance were found in the spinal cord and medulla only. The

Fig. 114.—Friedreich's Ataxia, Late Stage, showing curvature of trunk and deformities of legs.

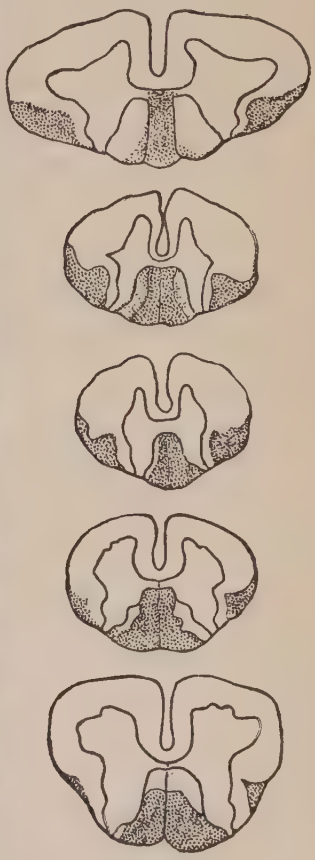


Fig. 115.—Showing the Distribution of the Sclerosis at Different Levels of the Cord in Friedreich's Ataxia.

cord was usually small, flattened, and apparently congenitally imperfect in development. In some cases two central canals have been seen. A sclerosis exists throughout the whole length

of the posterior and lateral columns, sometimes extending to the anterior columns (Fig. 115). The sclerosis is most marked in the postero-median columns, which are always affected *in toto*. The postero-external column is less involved, and there is often a narrow strip of healthy tissue between the posterior horn and the sclerosed area, also between the posterior gray commissure and the diseased parts. The posterior column sclerosis is usually most marked in the lumbar region. In the lateral columns the sclerosis always affects the crossed pyramidal tracts. The direct cerebellar tracts and the so-called ascending antero-lateral tract are diseased in some cases, but apparently not in all. In a few instances the anterior median columns are involved. A zone of healthy tissue is often found between the sclerosed pyramidal tracts and the posterior horn.

There are no important changes in the gray matter.

Some chronic leptomeningitis, especially on the posterior surface, has been noted. The medulla shows some traces of extension of the sclerosis, but the involvement of the cells of the hypoglossal nucleus is probably the most significant change. The brain exhibits no changes of importance in relation to the symptomatology of the disease. The posterior nerve roots are extensively sclerosed, the anterior roots less so, and the peripheral nerves show some degenerative changes.

It is thought by some (Déjérine) that the peripheral nerves are much less involved than in tabes dorsalis. It is also asserted that the sclerosis in the cord is really a neuroglia proliferation—a gliosis. Others assert that Friedreich's ataxia is a primary degenerative disease of the spinal vessels, and that the nervous tissue is secondarily involved.

Course and Prognosis.—The disease is a progressive one, though it may be stationary for a long time, and may even show temporary improvement. The longest period of duration of the disease on record is forty-six years and the shortest two years, the average being fifteen or twenty years. Death occurs from some intercurrent disorder.

Treatment.—A quiet life, good food, and favorable hygienic surroundings are the main therapeutic helps. Arsenic and various nerve tonics may be of temporary benefit. My cases and some of the French cases were benefited by suspension by the neck in a Sayre apparatus. If the disease appears in one member of a family, effort should be made to prevent its appearance in others. The infant should not be nursed by its mother; special care should be taken to prevent its getting any infectious fevers and to prevent it from receiving any falls or blows. Its life should be exceptionally quiet, so far as physical exertion goes.

Hereditary Ataxic Paraplegia.—There is a primary de-

generative disorder of the spinal cord occurring in children, and characterized by symptoms of ataxia with some cutaneous anæsthesia and spasm. The disease develops usually about the time of puberty. It progresses slowly, and takes upon itself the characters such as have been described under the name Ataxic Paraplegia. Such cases may for a time be considered of functional or hysterical character. A longer acquaintance with them, however, reveals the progressive and organic character of the disease.

Hereditary ataxic paraplegia belongs to the same group as Friedreich's ataxia. It is a primary degenerative disorder, involving, no doubt, the lateral and posterior columns of the spinal cord, but with a preponderance of the lesion in the lateral columns. The symptoms resemble those of ataxic paraplegia, but the progress is very slow. Cases have been described by Dr. Charles H. Brown.

Combined Sclerosis with Terminal Softening.

This is a rare disease, described by Dr. J. J. Putnam and later by myself. It is a rather rapidly progressive sclerosis of the cord, ending in a softening which may be due to tubercular or other secondary infection.

Etiology.—The patients are in most cases women. The ages range from forty-five to sixty-four years. In some there is a neurotic inheritance, and in several lead has been found in the urine at times. No distinct history of syphilis has been made out in any instance, nor are the patients alcoholic.

The disease runs a rather rapid course, varying from nine months to four years and averaging two years.

Symptoms.—The symptoms begin generally with numbness of the extremities, followed by progressive enfeeblement, and ending always in a paraplegia. Great emaciation and anæmia are present, and there is often an obstinate diarrhœa. No paralysis of any special groups of muscles occurs until the final paraplegia sets in. There are in some cases anæsthesia and ataxia, but spastic symptoms, with exaggerated knee jerk and ankle clonus, are the more common. Lancinating or girdle pains are very rare. The arms are affected, but less than the legs. The vision and other special senses and speech are not disturbed. Mental symptoms approaching dementia occur in the terminal stages in some cases.

The general course is that of a rather rapidly progressive affection causing paræsthesia and sometimes anæsthesia of the extremities, especially the lower, with progressive weakness of the extremities. This is associated with anæmia, general muscular emaciation, diarrhœa, ending in a paraplegia.

Pathological Anatomy.—The pathological appearances of the spinal cord as described by Putnam correspond to my own observations: in all the cases two sets of changes in the cord are recognizable, one of older date, consisting in a relatively dense sclerosis in the posterior columns and in the lateral column (mainly confined to the pyramid tracts); and one of subacute

character, and evidently of quite recent occurrence. This subacute process is, as regards the white columns, partly in new tracts, partly around the borders of the more dense sclerosis, and is chiefly characterized by the peculiar perforated appearance which indicates a somewhat rapid destruction of nerve tubes, with the œdematous distention or destruction of the intervening septa, associated with the formation of granule cells.

In the gray horns the degenerative change (partly recent, partly of older date) is indicated by a disintegration of nerve cells.

The *prognosis* is unfavorable.

The *treatment* is purely sustaining and symptomatic.

Spastic Spinal Paralysis, Hypertonic Paralysis, Spastic Paraplegia (Primary Lateral Sclerosis).

Definition.—Spastic spinal paralysis is a disease characterized by rigidity and weakness of the limbs, especially the lower, without muscular atrophy and with little sensory disturbance.

The disease is described here because it has always been called a "primary lateral sclerosis." No autopsy has as yet, however, proved that such a condition exists, and this name had best be dropped.

Etiology.—Spastic spinal paralysis must be considered simply a symptom-complex due to various causes. The principal ones are: 1, chronic dorsal myelitis in adults; 2, double cerebral hemiplegia in children; and, 3, a functional cause of unknown character. Rarer causes are multiple sclerosis and spinal tumor.

In adults the causes of spinal syphilis and myelitis are naturally the causes of this disease. In children the brain lesions of hemiplegia and Pott's disease are causes. The functional and curable form occurs oftener in men and is due to exposures, overexertion, or other influences, often perhaps of a specific character.

Symptoms.—The general character of the symptoms is that of a slowly developing paraplegia with excessive stiffness of the lower limbs. The reflexes are greatly exaggerated, clonic and tonic spasms occur, forming what is known as spinal trepidation or epilepsy. The muscles feel hard and do not waste; they may even be slightly hypertrophied. The electrical reactions are not notably changed. The gait is peculiar. The patient takes short steps, pushing the feet along, and the toes cling to the ground. There is a sense of weariness and numbness in the limbs, but rarely any anæsthesia or pain. The bladder and sexual functions are usually somewhat involved, and constipation occurs.

Later in the disease the arms may be affected, and in rare cases there is involvement of the trunk muscles. The cranial nerves are rarely reached, but sometimes an excessive irritability

of the muscles of mastication occurs, and a tap on the chin causes an abnormally quick spasmodic contraction or "chin jerk." In children there is rather less reflex irritability, but a greater tendency to contractures and deformities.

Course and Prognosis.—The disease, if organic, runs a very long course and has little tendency to shorten life. The ordinary forms are not curable, but their progress may be stopped and improvement occur. In the functional forms a cure may take place. I have observed it to do so in a few months.

The *pathological anatomy* is that of the diseases already mentioned. In dorsal myelitis there occurs a descending degeneration of the pyramidal tracts. The anterior cornual cells are cut off from the inhibitory influence of the brain, and hence the spasm. On the other hand, these cells in the lower part of the cord are not diseased, and hence there is no muscular atrophy. In double cerebral hemiplegia there is a descending degeneration from the brain along the direct and crossed pyramidal tracts.

The pathology of the functional cases is unknown; some of them may be of infectious or toxic origin, and the anatomical condition may be an exudative dorsal myelitis.

The *diagnosis* of the clinical type is easy. It depends on the existence of moderate paraplegia with spasm and exaggerated reflexes, without muscular atrophy or sensory disturbance. The special primary cause is often more difficult to determine. It is especially important to examine for Pott's disease, to exclude hysteria and amyotrophic lateral sclerosis. In Pott's disease there is usually pain, tenderness, and deformity. In hysterical paraplegia it is doubtful if there is ever true ankle or rectus clonus (Gowers). Besides, in hysteria the paralysis has a more acute onset and different course. In amyotrophic lateral sclerosis muscular atrophy soon appears. In multiple sclerosis evidences of sclerotic foci in the bulb and brain appear.

The chronic onset of the disease distinguishes it from hemorrhages, acute softenings, and injuries.

The functional form called by Bastian *hypertonic paralysis* is of rather acute onset and attended with severer symptoms of motor irritation. The weakness in the legs may be for a time very great, and profuse sweating sometimes occurs.

Treatment.—This must be directed to the cause. In syphilitic cases iodide of potassium and mercury are to be given. Warm baths, Turkish baths, massage, and galvanism are recommended. In my experience electrical currents of high tension, static sparks, and the secondary coil have been most useful, though they are condemned by some. Some patients feel better while standing, and they like work at a bench or counter. Gymnastic exercises, bicycle-riding, and orthopedic measures help children and very

chronic cases in adults. Arsenic, strychnine in quite small doses (gr. $\frac{1}{100}$), and the bromides sometimes give relief. Suspension is not often of value, and may do harm.

Secondary Degenerations of the Spinal Cord.

When any of the long-fibre tracts of the cord are cut across or destroyed, there soon results a degeneration. This extends

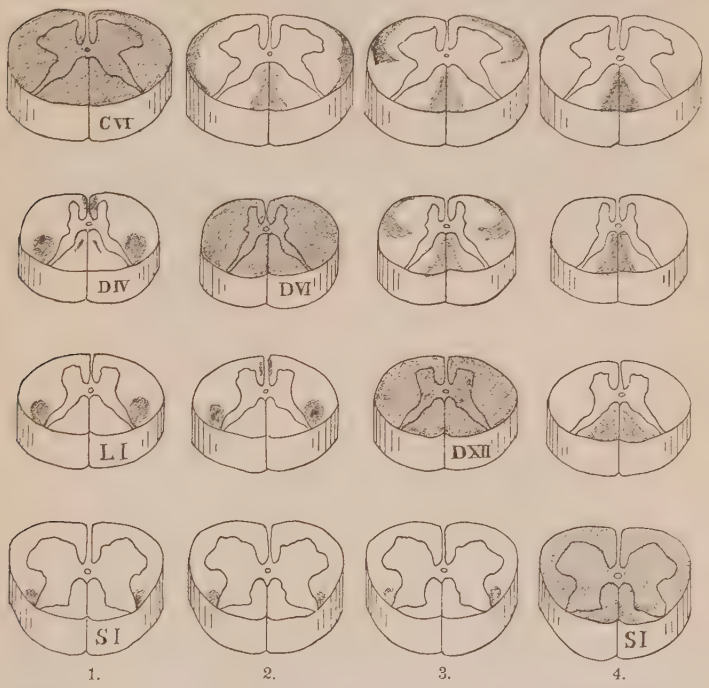


Fig. 116.—Showing the Tracts Affected in Secondary Degenerations of the Spinal Cord in Lesions at Different Levels. 1, Descending degeneration after lesion at sixth cervical; 2, ascending and descending degeneration, lesion at sixth dorsal; 3, ascending and descending degeneration, lesion at twelfth dorsal; 4, ascending degeneration, lesion at first sacral.

up or down in accordance with the direction in which the tracts carry impulses. Thus when the crossed pyramidal tract is cut across the degeneration extends down; when the column of Goll is involved it extends up. The degenerative process begins almost immediately and is complete in a few weeks. The myelin sheath swells, gradually breaks up and disintegrates; the axis cylinder is involved next. At the same time the connective tissue

proliferates and takes the place of the wasted nerves. Finally, long tracts of connective tissue have taken the place of the nerve tissue. The process may not be a complete one if the lesion does not entirely destroy the tract. The short-fibre tracts degenerate only a little way up and down (Fig. 116).

Secondary degenerations complicate and add to the pathological change in all organic diseases of the cord. In brain disease, involving the motor tract, as in hemiplegia, secondary degeneration extends into the cord and adds to the seriousness of the disease. Degenerations of the spinal cord, however, do not extend up to the brain except in the case of disease of the cerebellar tracts.

The accompanying figures show the extent of the secondary degeneration in total transverse lesions of the spinal cord at different levels. There is still some doubt as to the degeneration of the antero-lateral ascending tract, which some (Mott) assert is part of the cerebellar tract and goes to the vermis.

CHAPTER XIV.

THE PROGRESSIVE MUSCULAR ATROPHIES AND MUSCULAR DYSTROPHIES.

THE result of modern studies is to show that the anterior cornual cells of the spinal cord, the motor nerves and their terminal end-organs, the muscles, form a trophic unit, and that the same degenerative disease may attack either end or any part of this physiological mechanism. There is a clinical and pathological unity in all the different spinal and muscular types of atrophies.

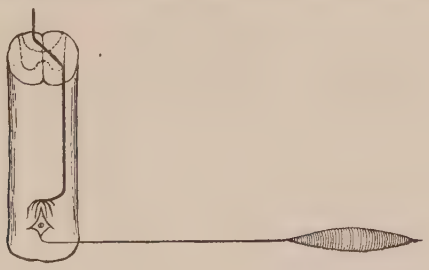


FIG. 117.—SHOWING: 1, Segment of spinal cord with anterior horn cell, end-brush, and lateral tracts, the parts affected in progressive muscular atrophies; and, 2, the muscle and its nerves, the parts affected in progressive muscular dystrophies.

But there are sufficient differences, also, to oblige us for convenience' sake to make certain classifications. Thus those disorders which attack chiefly and first the anterior horn cells and the pyramidal tract are called *progressive muscular atrophies;* those disorders attacking first the muscle tissue and its nerves are called *progressive muscular dystrophies* (Fig. 117). The progressive muscular atrophies of central origin may attack the motor nerve cells of the eye, of the throat and lips, of the upper or lower spinal cord. In accordance with the level affected the disease has received a different name. Sometimes the pyramidal tracts of the spinal cord are first and most involved. This has furnished excuse for another type. Then, again, while most cases of the muscular atrophy are acquired, there is one type of it which is a hereditary one. Thus we find the disease classified as follows.

Progressive muscular atrophies of central or nervous origin.	Progressive ophthalmoplegia.
	Progressive bulbar palsy.
	Progressive muscular atrophy (arm type).
	Progressive hereditary muscular atrophy (leg type).
	Amyotrophic lateral sclerosis.

The progressive muscular dystrophies have also been much subdivided, but they are essentially the same disease, as will be seen later.

Of the muscular atrophies, I have already described ophthalmoplegia. Beginning from above I will describe here bulbar paralysis, for though anatomically it belongs to disease of the brain, pathologically and clinically it belongs to the cord.

Glosso-Labio-Laryngeal Paralysis (Progressive Bulbar Paralysis).

This is a disease characterized by progressive wasting and paralysis of the muscles of the tongue, lips, palate, and throat, due to an atrophy of the nuclei of the nerves supplying those parts.

Etiology.—It is a disease of the degenerative period of life, most cases occurring after forty and between that time and seventy. It occurs rather oftener in men than women. A neurotic heredity is sometimes noted. Exposure to cold and excessive use of the muscles, mental strain, debilitating influences, lead, and syphilis are causal factors.

Symptoms.—The tongue is the part first affected. The patient speaks indistinctly and cannot articulate the lingual consonants *l*, *v*, *n*, and *t*. The tongue cannot be elevated and is protruded only a little distance. It looks scarred and wrinkled. The lips become weak and the patient cannot whistle nor make the consonants *p*, *b*, *m* or the vowel *o*. The saliva begins to dribble from the mouth. Disturbance in swallowing soon develops. Hard solids are taken with difficulty, next fluids, while semi-solids are generally managed best. The lips finally become so paralyzed that the mouth cannot be shut, and the lower part of the face is motionless and expressionless. The upper face wears an expression of anxiety and suffering, the saliva dribbles constantly, and the whole physiognomy of the patient becomes characteristic and pitiful in the extreme. The facial nerve may get somewhat involved. Articulation becomes almost entirely lost; the voice has a nasal twang from paralysis of the palate.

The patient has tired and uncomfortable sensations of dryness and stiffness about the throat. There is no pain or anæsthesia,

but occasionally there is impairment of the sense of taste. The throat reflex is usually lost, so that tickling it causes no reaction.

Electric irritability is at first unchanged, but in the later stages partial degeneration reaction occurs. In rare cases there is a rapid pulse and still more rarely glycosuria.

The laryngeal reflex becomes weak, the adductors also, but abductor paralysis is rare.

The mind is not affected, but there is often an emotional weakness and tendency to tears—not entirely unreasonable in view of the distressing nature of the malady.

The disease is often the terminal stage of spinal muscular atrophy; it may be associated with the latter, with amyotrophic lateral sclerosis or with ophthalmoplegia. All these types may occur together.

It runs a progressive course, with remissions of a few weeks or months. It lasts from one to three or four years. In one case it has lasted seven years.

The termination is eventually fatal. Death occurs through interference with swallowing, and inanition or a broncho-pneumonia or bronchitis may develop which ends the patient's life.

Pathology.—The primary lesion is found in the nuclei of origin of the hypo-glossal, glosso-pharyngeal, vagus, and spinal accessory nerves. The raphe fibres and the anterior pyramids are also usually somewhat involved. There is sometimes atrophy of the cells of the facial nerve and of the nucleus ambiguous, which is the motor nucleus of the vagus. The brunt of the disease falls, therefore, upon those more superficial or posterior nuclei which are representative of a continuation of the anterior cornual cells. If the disease is complicated with amyotrophic lateral sclerosis, or progressive muscular atrophy, or ophthalmoplegia, we find atrophy in the cord or ocular nuclei. The atrophic process is similar to that observed in the spinal disease.

The muscles of the tongue, and to a less extent of the orbicularis oris and throat, show evidences of degeneration and atrophy. In some cases the tongue is not shrivelled, owing to the presence of a fatty deposit, and on account of this the disease has been divided into atrophic and paralytic types, but this distinction is unnecessary. A few cases have occurred in which no bulbar lesion was discoverable, even by the microscope. In these cases the disease lay probably in the brain or peripheral nerves.

Diagnosis.—The disease must be distinguished from polioencephalitis inferior, bulbar apoplexy, tumors, and softening, from multiple sclerosis, and from chronic lesions of the cerebral hemispheres causing pseudo-bulbar paralysis. The slow onset, the progressive course, the bilateral character, the absence of involvement of sensory nerves, and the degenerative reactions are

always sufficient for a diagnosis. It is important always to note whether there is ophthalmoplegia and spinal muscular atrophy associated with the disease.

Treatment.—The patient should be kept quiet; he must be overfed and given massage and electricity in moderation. The same drug treatment as in the spinal disease is indicated. Small doses of morphine, gr. $\frac{1}{24}$ to $\frac{1}{36}$, and of atropine may be given also. Electricity should be tried for a short time twice or even thrice daily, if possible. The faradic current may be used, alternating or combined with the galvanic. Galvanization of the neck and medulla appears to do no good. After a time it may be necessary to feed with a tube or even to do tracheotomy.

Progressive Muscular Atrophy (Progressive Spinal Amyotrophy; Duchenne-Aran's Disease).

This is a disease characterized by a slow, progressive muscular atrophy of the muscles of the extremities and trunk, with consequent paralysis, not accompanied with any notable sensory disturbance, and due to a progressive atrophy of the motor and trophic cells in the spinal cord.

Etiology.—The disease affects persons in the middle period of life (twenty-five to forty-five). The extremes are fourteen and seventy years (Gowers). It is more frequent in males. Heredity is rarely, if ever, a factor. Great mental strain, exposure, traumatism, excessive use of certain groups of muscles, acute infectious diseases—especially typhoid, measles, cholera—child-birth, acute rheumatism, syphilis, and, more than anything else, lead-poisoning are causes. It may complicate locomotor ataxia. The causes, as may be seen, are much the same as those of bulbar paralysis.

Symptoms.—The patient suffers at first from slight rheumatoid pains in the shoulder or arm, associated with some feelings of numbness and weariness. Muscular wasting then begins to appear, and usually in one hand. The adductor longus pollicis is very early affected, also the thenar muscles and the interossei. The atrophy spreads from muscle to muscle, and does not follow the distribution of nerves, although the ulnar nerve supply is most seriously disordered. The ball of the thumb becomes flattened, and the patient cannot abduct or flex it well. When the radial interossei are reached the forefinger cannot be abducted, and this is often an early sign. The disease gradually extends upward, attacking the flexors and extensors of the forearm, then the upper arm and shoulder. Meanwhile the hand has become thin and flattened, flexion of wrist and extension of fingers are lost, and a characteristic "griffin-claw" appearance results. Mean-

while (three to nine months) the other arm begins to be affected. Occasionally there is a remission for a time.

In a few cases the atrophy begins first in the shoulders and arm, attacking the deltoid, biceps, and triceps, then extending downward to the hands. This constitutes the "upper-arm type."

If, as is usually the case, the disease continues to progress, it passes from the shoulder girdle to the deep muscles of the back, then downward, involving successively the hip and thigh muscles, the glutei, the crural extensors and abductors being oftenest chosen. The leg muscles may be finally involved, but they usually escape. The disease as it descends continues its progress in the trunk, involving the intercostals. It slowly ascends the neck also, and finally leads to paralysis of the diaphragm, or a bulbar palsy may set in.

It will be seen that the ordinary course of the disease is from the lower-arm-muscle groups (ulnar and median) up to the shoulder group (middle cervical nerves), then down through the dorsal and lumbar nerves, rarely reaching the sacral groups. In very rare cases it begins in the legs and ascends.

Along with the wasting there is a corresponding weakness and paralysis, but the paralysis is the result of the atrophy and does not precede it. Fibrillary twitchings of the muscles occur; the idiopathic muscular contraction caused by striking it a blow is very marked; myoid tumors are easily brought out. In some cases the muscles are flaccid and toneless, and the deep reflexes, knee jerk, and arm jerk disappear early (atonic atrophy), but in other cases the rigidity and tonicity of the muscles are increased, the knee jerks exaggerated, and we have tonic atrophy. This condition may be so marked as to give rise to a special clinical type of progressive atrophy known as *amyotrophic lateral sclerosis.*

The electrical irritability of the muscles lessens to both galvanic and faradic currents, but no marked qualitative changes occur at first. Eventually we may get partial degeneration reactions, but these occur late in the disease, unless this runs a very rapid course, when fairly typical degeneration reactions may be got. A peculiar contraction of the upper limbs is sometimes produced by placing the negative pole of the galvanic battery over the fifth cervical vertebra, and the positive in the triangle just below the lower jaw (diplegic contraction of Remak). A peculiar palmar spasm is described by Voeter, caused by suddenly interrupting a faradic or galvanic current passed along the affected arm.

In typical cases of progressive muscular atrophy there is no anæsthesia; and when such symptoms develop, the presence of peripheral disease or of syringo-myelia or spinal tumor must be expected. The patients may suffer from rheumatic-like pains and from paræsthesiæ.

The affected parts often show excessive sweating and congestion and evidence of vasomotor disturbance. This may involve the face on one or both sides; one pupil may be larger than the other, due to irritation of the cilio-spinal centre. The iris-reflex, however, is preserved, and the optic nerve is never involved.

The sexual power is often weakened, but the sphincters are not attacked. The urine shows variations in the amount of urea. There is usually an increase of lime salts.

Complications.—The most common complication is an extension of the process to the medulla, causing disturbance of speech and swallowing. Muscular atrophy complicates locomotor ataxia, but is rarely complicated by it. A high degree of spasm and rigidity of the legs, particularly, may occur, causing the condition known as amyotrophic lateral sclerosis.

Course and Duration.—The disease usually progresses steadily until it has reached an advanced stage, when it may stop. Remissions may occur earlier, however, and even some improvement take place; the disease then ordinarily progresses again. It lasts from two years to thirty or more, but on the average not over ten or twelve years. Death usually occurs from pulmonary disease, owing to the weakness of the respiratory muscles. Sometimes the extension to the medulla and involvement of the muscles of deglutition and of the larynx is the cause of death.

Pathology.—The primary anatomical change is a degenerative atrophy of the cells of the central parts and anterior horns of the gray matter of the spinal cord. The atrophy gradually extends and involves the whole anterior horn. It also extends vertically, first down, then up. Along with this atrophy are degenerative changes in the lateral columns; consecutive to this there is atrophy of the anterior roots, peripheral nerves, and the muscles. The disease begins in the deeper parts of the anterior cornua, involving the central and median groups of cells. These are more concerned in nutrition and in the finer muscular movements of the extremities. Hence atrophy always precedes, or at least keeps pace with, paralysis. The levels affected are the lower cervical and upper dorsal; but if the disease is extensive, the dorsal, lumbar, and sacral cord are also involved. The affected part is nearly free from nerve cells, and those present are atrophied, their processes are short or absent, and the cell has lost its angular appearance. Sclerotic and pigmentary changes are observed. The neuroglia and connective-tissue cells are increased in number, but there are no marked changes in the blood-vessels, though these may be much dilated. There is always some degeneration of the lateral columns, and this may be very complete. It is confined chiefly to the pyra-

midal tracts, but extends somewhat anteriorly into the mixed lateral column. It does not affect the cerebellar or ascending lateral tracts. The degeneration has been traced up into the brain as far as the internal capsule and even to the cortex. The anterior columns may be slightly affected. The posterior horns, columns, and roots are normal.

The affected muscles show various degrees of degeneration. They are pale and streaked with yellow due to fatty deposits. Some fibres may be simply narrow and shrunken, others have lost their striation and become granular from deposit of fat globules or degenerated muscle elements. Other fibres have lost their striations and look as if filled with a homogeneous, glassy-looking substance containing a few fat granules (vitreous degeneration); others show a longitudinal striation. The interstitial connective tissue is increased and in places has taken the place entirely of the muscles. The capillaries and small vessels are distended. Healthy fibres may be seen among the diseased. Changes have been found in the sympathetic nervous system, but they are unimportant.

The *diagnosis* has to be made from the progressive muscular dystrophies, chronic poliomyelitis anterior, syringo-myelia, neuritis, and neuritic family atrophy.

In the muscular dystrophies there is commonly a history of heredity; the disease begins usually in childhood or adolescence. It attacks the lower limbs oftener; it is slower in progress; there are no fibrillary contractions, and the degeneration reaction does not occur.

Chronic poliomyelitis anterior begins suddenly and, having reached its height, does not progress, but remains stationary or improves. The paralysis occurs first, the wasting follows. It affects groups of muscles physiologically related, while progressive muscular atrophy attacks muscles only anatomically related. There are cases, however, which seem to be on the border line between the two diseases.

Syringo-myelia is distinguished by the presence of peculiar sensory and trophic disorders.

Neuritis caused by lead-poisoning is detected by the history of the case, its tendency to affect the extensors of the arm chiefly, and the absence of a progressive tendency. Sometimes, however, lead-poisoning and palsy end in true progressive muscular atrophy.

Ordinary multiple neuritis is distinguished easily by its rapid onset and the presence of painful symptoms.

The hereditary or "leg type" of progressive muscular atrophy is characterized by its beginning in the legs, by a good deal of sensory disturbance, typical degeneration reactions, and hereditary or family history.

Treatment.—The patient should be well fed and have rest, quiet, and fresh air. Careful local faradization and galvanization of the spine and neck are indicated. Massage does no good. Hypodermic injections of strychnine in the affected member, gr. $\frac{1}{80}$ to $\frac{1}{30}$ daily, the internal use of arsenic, phosphorus, iron, quinine, and cod-liver oil sometimes are beneficial.

In a few cases with a syphilitic history, mercury and iodide of potash have proved useful. The essentials of treatment are rest, electricity, strychnine locally, the administration of powerful tonics, and over-feeding. Nitroglycerin, morphine, atropine, nitrate of silver, chloride of gold and of barium, and the nitrate of uranium may be tried.

Progressive Hereditary Muscular Atrophy of Leg Type (Charcot-Marie Type).

This is a hereditary or family muscular atrophy of central (or neuritic?) origin, beginning in the legs and extending upward. It affects males more than females, but the difference is not great. It almost always begins before the age of twenty. It attacks first the muscles of the leg, not the foot, involving the peronei, then the extensors of the toes, then the calf muscles. The thighs escape till later. After some years the upper extremities and small hand muscles are reached. The shoulder and arm, neck and trunk muscles escape. There are occasionally fibrillary contractions; and always partial or complete degenerative electrical reactions. The patients complain of some pain and numbness, but there is no anæsthesia.

The disease runs a long course, with remissions, and resembles in prognosis the dystrophies. The outlook is better than in the arm type, but the disease is not curable.

Some authorities assert that the disease is due to a progressive degenerative neuritis. In the writer's opinion the anterior horns of the spinal cord are primarily attacked.*

The *treatment* is the same as for the other forms of hereditary muscular atrophy.

Amyotrophic Lateral Sclerosis.

This is a form of progressive muscular atrophy associated with spastic symptoms and paralysis. It ends in or may be early associated with bulbar paralysis or even with ophthalmoplegia.

The *etiology* resembles that of ordinary forms of spinal muscular atrophy.

* The writer has seen the disease in a typical form in one member of the first generation, in two members of the second. A child of one of the latter had, at the age of two years, a typical attack of anterior poliomyelitis.

The *symptoms* of typical cases such as were first described by Charcot are those of a slowly developing paralysis. This affects the upper extremities first and spreads diffusely. The atrophy is less marked than the paralysis. There is rigidity, exaggerated reflexes, and sometimes tremor. Fibrillary contractions are present. There are no notable sensory symptoms. The legs are affected later; the paralysis and spastic condition of the legs are antecedent to the atrophy. The visceral centres are not involved. The disease progresses rather rapidly and finally involves the medulla, causing bulbar paralysis and death. Sometimes ophthalmoplegia is also present.

In less typical cases the disease begins with spinal and bulbar symptoms almost simultaneously. In a case under the writer's observation there was ophthalmoplegia, bulbar paralysis, and spinal atrophy beginning at nearly the same time. Ordinary cases of spinal muscular atrophy may also be early associated with rigidity and exaggerated reflexes. The jaw jerk is often increased in this disease, which cannot be said to be really distinct from progressive spinal muscular atrophy; it runs usually a rapid course, causing death in two or three years.

THE PROGRESSIVE MUSCULAR DYSTROPHIES.

This name is given to various forms of progressive muscular atrophy in which the disease is hereditary in character and attacks primarily the muscles. Although a muscular disease, yet its close relationship with spinal muscular atrophies leads us to describe it here.

A number of types have been described, the distinctions being based chiefly on the part of the body first affected. These types are not of great importance, but may be enumerated here for convenience:

1. Pseudo-muscular hypertrophy.

(*a*) Leyden-Möbius or hereditary type, appearing in children, beginning in back and lower limbs.

2. Erb's juvenile type, or scapulo-humeral type, beginning in childhood or youth, usually in the shoulder girdle or trunk.

3. Landouzy-Dejerine type, or infantile progressive muscular atrophy of Duchenne, or facio-scapulo-humeral type. It resembles the preceding form, with the exception that it involves the face.

4. The peroneal or leg type has been classed with the dystrophies, but is probably of spinal or neuritic origin, and has been described with the atrophies (see page 268).

The essential unity of all these different forms is shown by the fact that cases occur in which pseudo-hypertrophy takes place in the scapulo-humeral and other types, by the fact that a dis-

ease resembling pseudo-hypertrophic paralysis occurs without any hypertrophy, and by the fact that different types occur in the same family. The unity of the spinal and muscular forms is shown by the same kind of clinical evidence.

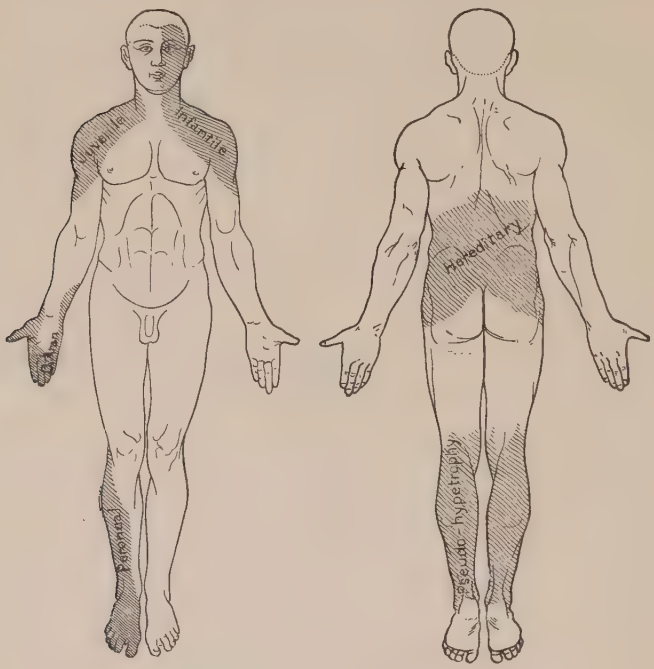


FIG. 118.—SHOWING THE PARTS FIRST ATTACKED IN THE DIFFERENT TYPES OF MUSCULAR DYSTROPHY AND MUSCULAR ATROPHY. The shaded parts in A show the place of onset of progressive muscular atrophy of ordinary or Duchenne-Aran type, of leg type, and of types 2 and 3 in text. B shows place of onset of types 1 and (*a*) in text.

At the same time the classical types of dystrophies are very different clinically from the spinal amyotrophies and hence must be separately described. The differences will be shown under the head of diagnosis.

PSEUDO-MUSCULAR HYPERTROPHY (ATROPHIA MUSCULORUM LIPOMATOSUS).

This is a disease beginning in childhood and characterized by a progressive weakness of the legs associated with an apparent muscular hypertrophy due to a deposit of fat in the wasting muscles.

Etiology.—The disease attacks boys much oftener than girls. It begins, in the vast majority of cases, under the age of ten, often at the close of infancy, very rarely not till after puberty. Heredity is a very important factor (in three-fifths of cases), the hereditary influence being almost always transmitted by the mother. A psychopathic or neuropathic condition is often found in the ancestry. Neuroses, syphilis, intemperance, consanguinity, are not factors in hereditary causation. Injury and an acute disease sometimes appear to act as exciting causes.

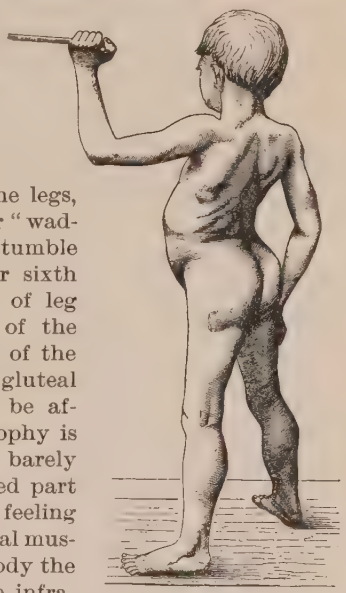


Fig. 119.—Pseudo-Muscular Hypertrophy (Erb).

Symptoms.—The first symptom noticed is a weakness in the legs, which shows itself in a peculiar "waddling gait" and a tendency to stumble and fall. A little later (fifth or sixth year) an apparent hypertrophy of leg muscles, particularly of those of the calves, develops. The extensors of the knee or one of them and the gluteal and lumbar muscles may also be affected. Sometimes the hypertrophy is very great, at other times it is barely noticeable (Fig. 119). The affected part has a peculiar, hard, non-elastic feeling to the hand, not like that of normal muscle. In the upper part of the body the hypertrophy oftenest attacks the infra-spinatus. The supra-spinatus and deltoid may be somewhat involved. The lower parts of the pectoralis major and latissimus dorsi are also usually atrophied, giving a characteristic appearance to the shoulders. The upper-arm muscles are often slightly wasted, the forearm, neck, and face rarely. The tongue may be hypertrophied.

Along with the pseudo-hypertrophy there occurs an atrophy of certain groups of muscles; and after a time the pseudo-hypertrophy disappears and an atrophy takes its place. In the lower limbs the muscles most atrophied are the flexors of the hips, then the extensors of the knee and those of the hip. The calf muscles fail before the anterior tibial. The atrophy and consequent weakness of the lower-limb muscles causes great difficulty in going upstairs, the gait becomes more waddling, and the patient loses the power of getting up when lying on the floor. These peculiarities are due chiefly to the weakness in the exten-

sors of the knees, the extensors of the hip, and the flexors of the hip. By reason of the same defects, the child when standing has an antero-posterior curvature of the spine with the concavity backward (lordosis). This is due to the weakness of the extensors of the hips, which, acting from the hips, are unable to tilt the pelvis back. On sitting this lordosis disappears, and is replaced often by a curve in the opposite direction due to weakness of the erectors of the spine. There may be some lateral curvature also. In consequence of the weakness and contractures of the leg muscles, there early develops a talipes equinus, and later the legs may become flexed on the hips and the forearms on the arms.

The muscles show no fibrillary twitchings and rarely any degenerative reactions, but there is sometimes a peculiar tetanic contraction with both the faradic and the galvanic current.

The knee jerks and elbow jerks gradually weaken and in time are lost.

There is no pain or other disturbance of sensibility.

The affected parts feel cold and look reddened as if from deficient vasomotor innervation. The organic spinal centres are not involved. Intelligence is usally good.

Course.—The disease runs a chronic but variable course. Its progress is at first slow; after walking becomes impossible it may progress. It lasts from ten to twenty-five years. In a few cases patients have reached the age of fifty or sixty years, even when the disease began in youth. The earlier the disease begins the more rapidly it extends; the more pronounced the tendency to lipomatosis, the more rapid is the course.

Pathological Anatomy.—This disease, like the other forms of dystrophy, is a degenerative one, the process affecting first the muscle fibres; the connective tissue and terminal nerve filaments being secondarily involved. In the same patient and even the same muscle the most varied changes may be noted. In the early stages there is a true hypertrophy of some of the fibres, a condition thought to be characteristic of the muscular dystrophies in distinction from the spinal atrophies. Besides swelling and hypertrophy of fibres, one sees atrophy of the fibres; the bundles are rounded; there is increase of muscle nuclei, splitting of fibres, vacuolization, and tendency to break up into fibrillæ (Erb). The connective tissue at first shows evidence of irritation and proliferation. Finally, as the muscular atrophy progresses, connective tissue increases and takes its place, until a dense, hard myo-sclerosis results. In some parts there is deposit of fat in the connective-tissue cells, and this may increase until an extensive lipomatosis exists. In the later stage of the disease the fat deposits are absorbed and there is only atrophied muscle and connective tissue. The nerves and spinal cord are usu-

ally normal; when changes are found they are secondary to the muscular disease (Fig. 120).

The process is then, first, hypertrophy of muscle fibre and increase of muscle nuclei, swelling and rounding of fibres, and spilling of the same; then increase of connective tissue, with corresponding atrophy of muscle and deposit of fat.

The process is a primary degeneration due to an inherent nutritional weakness of the muscle. In a measure it is true that

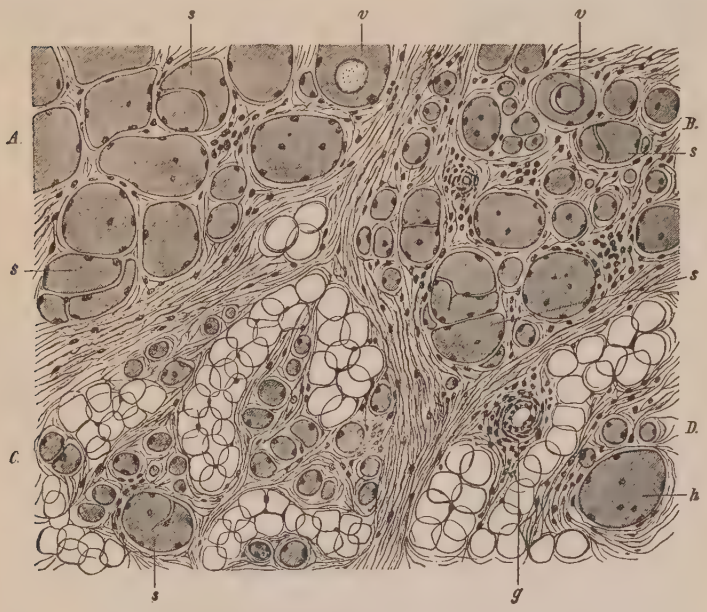


FIG. 120.—PARTIALLY DIAGRAMMATIC, showing A, hypertrophied fibres; B, mixture of hypertrophy and atrophy; C, D, atrophy and fatty deposit; *v*, vacuolization; *s*, splitting; *h*, hypertrophy of fibres; *g*, thickened blood-vessel (Erb).

those muscles embryologically latest developed are earliest attacked.

2. The *juvenile form of Erb* or scapulo-humeral form of dystrophy begins in childhood or early youth, a little later than pseudo-hypertrophy. The shoulder girdle is first affected, later the arm. The forearm and legs are attacked very late. Part of the pectorals, part of the trapezii, latissimus dorsi, rhomboid, upper-arm muscles and supinators are affected, while the supra and infra spinati and forearm and hand usually escape. There may be true and false muscular hypertrophy. There are no fibrillary contractions or degenerative reactions.

3. The *facio-scapulo-humeral form* or infantile progressive muscular atrophy begins in early childhood (third or fourth year) usually, but may develop late. The atrophy attacks first the face, giving a characteristic appearance known as the myopathic facies. There is a weakness of the oral muscle, which causes the lips to protrude and produces a symptom called the "tapir-mouth." The atrophy respects the eye muscles as well as those of mastication and deglutition. It extends to the shoulders and arms next, then it pursues the ordinary course of the dystrophies.

Fig. 121. Fig. 122.

Fig. 121.—Muscular Dystrophy having Characters of Juvenile Type (2) and Pseudo-Hypertrophic Type (1*a*) (Putzel).

Fig. 122.—Juvenile Type, showing atrophy in shoulder and arms (Erb).

Prognosis.—The patient never recovers, but the disease sometimes comes to a standstill and there may even be some improvement, especially in cases beginning late.

Treatment.—The prophylaxis is important. It consists in preventing the marriage of women belonging to dystrophic families; if a dystrophy has developed in one child, it would be unwise to take the risk of bringing others into the world. Or if children are already born, they should receive the most careful nourish-

ment, out-door life should be secured, and the dangers from trauma and the infective diseases be prevented. Infants should not be suckled by the mother if she belongs to the dystrophic family.

The moderate use of massage and gymnastics is very important and useful. All kinds of tonic measures are indicated, such as cold baths, good nourishment, arsenic, strychnine, and phosphorus and fats. Tenotomy and other orthopedic measures may be useful in the later stages.

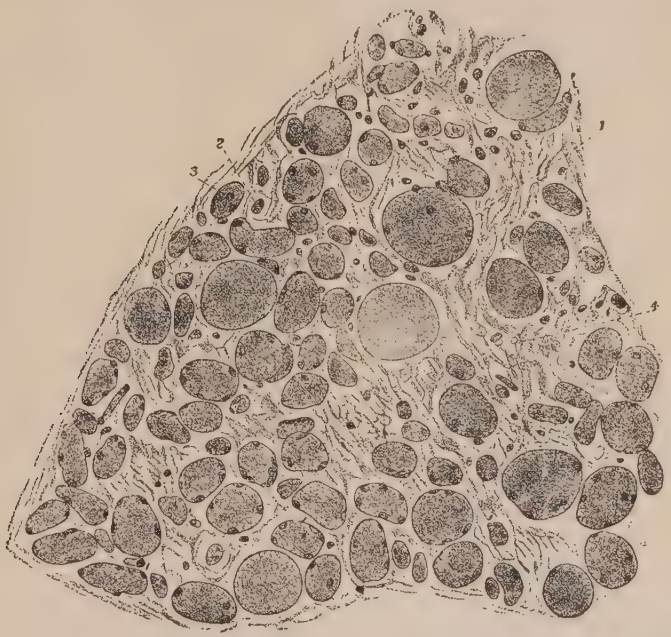


FIG. 123.—SHOWING CHANGES IN ARTHRITIC MUSCULAR ATROPHY. 1, Normal fibre; 2, atrophied fibre; 3, vacuolated fibre; 4, connective-tissue proliferation (Darkschewitch).

ARTHRITIC MUSCULAR ATROPHY.

In inflammation of joints the muscles moving are affected by a simple atrophy which is called arthritic.

Etiology.—Rheumatic arthritis is the commonest cause.

Symptoms.—The shoulder-girdle muscles are oftenest affected. Whatever the joint, it is the extensor muscles which are first and most attacked; the muscles above the joint are more susceptible to the atrophy than those below. The atrophy is rather rapid in the first few weeks and then becomes slower. The muscles

affected waste throughout their whole length. They show no fibrillary contraction and no degenerative electrical reaction. There is often an increased irritability, so that an exaggerated tendon reflex or even clonus may be produced. There is no pain or tenderness or anæsthesia in the muscles.

Pathology.—The anatomical change is a simple atrophy and shrinking of the muscle fibres, with some increase in muscle nuclei, little vacuolation, no swelling or splitting of fibres (Darkschewitch—Fig. 123). There is some increase in interstitial tissue, but this is slight. The nerves and spinal cord are normal.

The atrophy is probably due to reflex action. It is a reflex tropho-neurosis. Even a neuralgic joint may cause an atrophy.

The *prognosis* is good. If the arthritis gets well the muscles are also restored.

The *treatment* consists of electricity and gentle massage and exercise. Internal treatment must be directed to the arthritis.

Occupation Muscular Atrophies.

As a result of constant over-use muscles sometimes atrophy. This applies especially to the smaller muscles of the hand. Thus there occurs an atrophy of the thenar eminence in lapidaries and in persons who constantly use this group of small muscles.

The biceps sometimes wastes in smiths, and I have seen atrophy of the calf muscles in a ballet-dancer.

The disease does not tend to extend beyond the muscular group first attacked. Its seat is probably the muscles themselves. The treatment is rest.

CHAPTER XV.

TUMORS AND CAVITIES OF THE SPINAL CORD.

Etiology.—Tumors occur rarely in the spinal cord. The largest tables of cases include less than one hundred. The commonest age is thirty to fifty; tubercle occurs earlier (fifteen to thirty-five —Herter) and lipoma is congenital. Males are more subject than females. Tuberculosis, syphilis, and cancer predispose to the disease. Injuries and exposures appear sometimes to excite the growth of spinal neoplasms.

Symptoms. — These vary with the location, character, size, and rate of the growth of the tumor. No definite clinical picture can be drawn. The symptoms are such as result from a foreign body slowly and progressively irritating and destroying the roots and substance of the spinal cord. Pain appears early and is very constant, continuous, and severe. It is generally referred to nerves running out from the cord in the region of the tumor; a girdle sensation is felt. Numbness, hyperæsthesia, and later anæsthesia occur. Tenderness over the spine and rigidity are not very frequent. The sensory symptoms are usually more on one side, but may become bilateral. Spasm, contracture, and exaggerated reflexes usually soon develop, involving

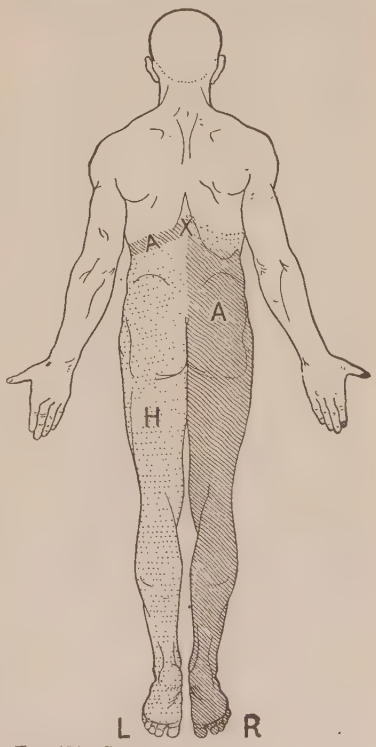


FIG. 124.—SHOWING THE CONDITION IN A BROWN-SÉQUARD PARALYSIS DUE TO A TUMOR GROWING IN THE LEFT SIDE OF THE SPINAL CORD. On the left side, hyperæsthesia, ataxia, paralysis, exaggerated reflexes. At the upper limit is a band of anæsthesia. On right side, anæsthesia.

one or both legs or an arm and leg. Later paralysis, atrophy, loss of control of the bladder and rectum, and bed-sores follow, and death ensues from exhaustion.

When the disease is cervical the four extremities and trunk muscles may be gradually involved, and there is rigidity of the neck and optic neuritis. If lower down, there develops a hemiparaplegia, later a complete paraplegia, usually with exaggerated reflexes. If the tumor is in the lumbar region the reflexes are sooner lost and the sphincters early involved.

A rather frequent type of symptoms caused by spinal tumors is that known as a *Brown-Séquard paralysis* or hemiparaplegia. In a typical case of this kind there is paralysis of motion and muscle sense on the side of the lesion, paralysis of cutaneous sensation, especially of pain, and temperature on the opposite side. On the side of the lesion the temperature may be slightly raised;

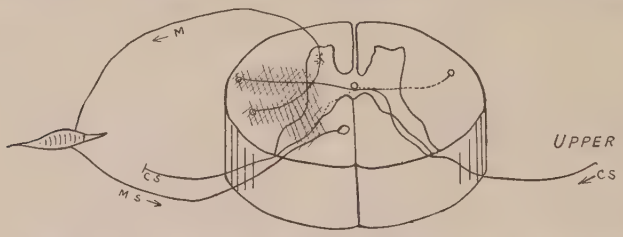


Fig. 125.—Showing the Mechanism of the Production of a Brown-Séquard Paralysis. The shaded part on the left represents the lesion. This involves the pyramidal tract and motor fibres M, causing paralysis and spasm on same side; also the muscle sense nerves M S on same side and the cutaneous sensory nerves C S of opposite side.

there is often hyperæsthesia, and reflex action is increased. There may be a band of anæsthesia at the level of the lesion and on the same side (Figs. 124, 125).

The symptoms vary according as the tumor is outside or inside the dura. The common extra-dural forms are lipoma, cancer, gumma, and sarcoma. The greater amount of motor and sensory irritation, the evidence of some vertebral disease, existence of malignant tumor elsewhere, the absence usually of hemiparaplegia, characterize extra-medullary tumors. The common forms of intra-dural or medullary tumor are glioma and tubercle. In these cases pain and spasm and rigidity are rather less common in the early stage; hemi-paraplegia is more common. A secondary myelitis sometimes develops.

The duration of the disease ranges from three months to five years, the average being two or three years.

Pathology and Pathological Anatomy.—All forms of tumor occur, but the commonest are gliomata and sarcomata, and after

this fibromata, myxomata, gummata, and tubercles. Cancer is rare; echinococcus and cysticercus are the only parasitic tumors found. The majority start from the membranes, next from the cord, and fewest from the vertebræ.

The meningeal tumors are mostly sarcomata and their various modifications, fibroma, enchondroma, carcinoma, and lipoma. The myelonic or intra-spinal tumors are commonly gliomata, sarcomata, tubercle, and syphilomata.

Spinal tumors are small in size, ranging from one-fifth to one and one-fifth inches (one-half to three centimetres) in diameter. The glioma may diffuse itself for a long distance through the centre of the cord, forming cavities (syringo-myelia). The sarcomata may likewise be irregularly spread along the surface of the cord. Spinal tumors are usually single, but fibromata, the parasites, and sarcomata may be multiple.

The favorite locations for spinal tumors are just below the mid-cervical, the upper and the lower dorsal regions. The secondary changes produced by tumors are softening, hemorrhages, secondary degenerations, and inflammatory reaction. The origin and histology of spinal tumors does not differ from that of tumors elsewhere.

FIG. 126.—SARCOMA OF SPINAL CORD, MID-CERVICAL REGION.

The Diagnosis.—The disease has to be distinguished from vertebral caries, transverse myelitis, and hypertrophic pachymeningitis. The points to be noted as regards caries are the absence of an external tumor or kyphosis; the small degree of tenderness and rigidity, the age, and absence of tubercular diathesis. The progressive course, beginning with pain, followed by motor and then sensory paralysis, and the localization of the symptoms excludes myelitis. The differentiation from hypertrophic pachymeningitis is often very difficult.

The character of the tumor cannot often be determined certainly. The probabilities are in favor of sarcoma or glioma, especially in middle life. Syphiloma may be suspected from the history and results of treatment. Tubercle is very rare.

The *prognosis* is bad except in syphiloma, and even here it may be serious. Tubercle may perhaps cease to grow. Surgical interference now saves the life of some cases.

Treatment.—In syphilitic tumors appropriate remedies may do good and should be vigorously used. In tubercle, tonic treatment, iodine, and cod-liver oil may be of service. In gliomata and sarcomata, nitrate of silver and arsenic may be tried. In other forms, symptomatic treatment is all that can be recommended medically. In all cases of spinal tumor surgical interference should be considered; extra-dural tumors, if taken early, can be removed with great benefit. Even in medullary tumors enucleation may do some good in the early stage. Exploratory operations are justifiable if there is no question as to the diagnosis of tumor. The mortality from such operation in capable hands is very low, though it is more dangerous than trephining the skull.

CAVITIES IN THE SPINAL CORD.

The cavities of the spinal cord are known as

1. Hydromyelia.
2. Myelitic cavities.
3. Syringo-myelia.

There may be various combinations of these processes, the most common being a combination of hydromyelia and syringomyelia.

Hydromyelia.

This is a dilatation of the central canal of the cord, the cavity being filled with fluid. The condition may be associated with hydrocephalus or with spina bifida, or it may be independent of these conditions. The dilatation may be cystic and irregular or, as is more usual, extend throughout the cord. The dilatation extends more posteriorly, because the posterior columns are formed latest. The abnormality may be slight and give rise to no symptoms. Or a pathological process like a gliomatosis may develop upon it.

Myelitic and Hemorrhagic Cavities.

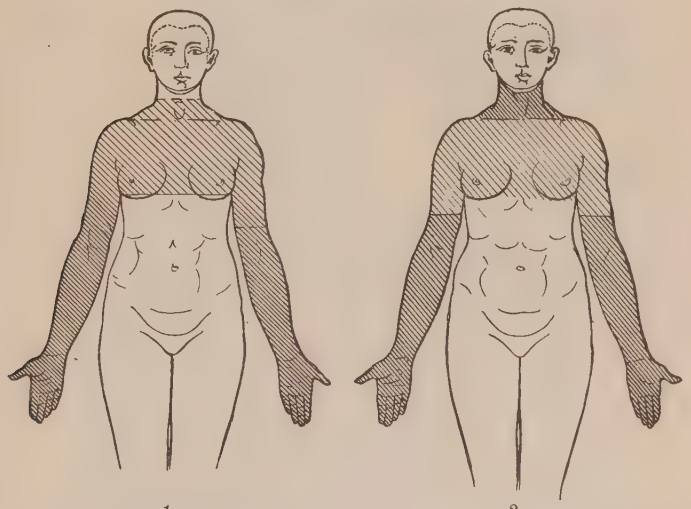
Such cavities may be formed in the cord by a central excavating myelitis or by small hemorrhages. These cavities are usually small, irregularly distributed, and are lined with connective tissue. They may be formed in rare cases in connection with hydromyelia or neoplasms.

Syringo-Myelia.

Syringo-myelia is a disease of the spinal cord characterized by a development of gliomatous tissue in the central parts, with formation of cavities. Clinically the disease is sometimes characterized by peculiar disturbances of sensation and nutrition. In many cases, however, the symptoms are atypical and the disease cannot be recognized during life.

The description here given corresponds to the classic manifestation of the disease, and it is intended to refer to syringomyelia produced by gliomatosis only.

Etiology.—The disease is rare, about two hundred cases having been reported. It is more frequent than amyotrophic lateral



1. 2.

Fig. 127.—Distribution of Cutaneous Anæsthesia in Syringo-Myelia. 1 shows area of analgesia; 2 shows that of thermo-anæsthesia. The darker shades show where there is anæsthesia to pain and temperature (Bruhl).

sclerosis and rather less frequent than multiple sclerosis. It occurs oftener in men than women, and develops early in life, between the ages of fifteen and twenty-five.

It occurs especially in persons who follow manual occupations such as butchers, tailors, etc. Traumatism, pregnancy, and infectious diseases seem occasionally to give rise to it. Heredity, syphilis, and alcohol are not causal factors.

Symptoms.—The disease begins insidiously with some aching pains in the neck and arms and paræsthesia of the hands. There is soon a muscular atrophy of the hands resembling spinal progressive muscular atrophy, with perhaps anæsthesia. As the disease progresses the weakness and atrophy of the hand muscles become more noticeable and gradually extend toward the trunk. The atrophy comes on in both extremities at about the same time.

Fibrillary contractions and partial degeneration reaction may be observed. Cutaneous anæsthesia of the affected hand and arm to temperature and pain, but not to touch, takes place; and this is so marked as to be almost pathognomonic of the disease (Fig. 127).

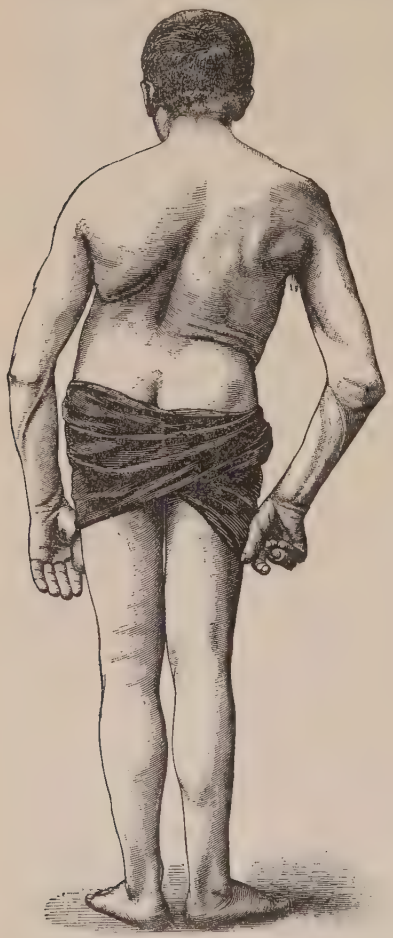


Fig. 128.—Showing Curvature of Spine in Syringo-Myelia (Erb).

The legs do not become affected until late, and then generally show a spastic paraplegia. The throat and face are rarely involved. There is a scoliosis of the spine, generally in the dorso-lumbar region (Fig. 128).

Vasomotor, secretory, and trophic symptoms are prominent. The hands may be œdematous or red and congested. Sweating or dryness of the skin may occur. Eruptions appear on the skin, such as bullæ, herpes, and eczema. Painless whitlows attack the fingers and may destroy the terminal phalanges. Erosions and ulcerations also occur. The nails become dry, brittle, and drop off (Fig. 129). Arthropathies and spontaneous fractures have been observed. The pupils may be unequal and the bulb retracted.

Late in the disease symptoms of involvement of the medulla develop. At this time also the bladder, rectum, and genital centres are attacked. The disease progresses slowly for years, with remissions of various degree.

The cardinal symptoms are a progressive muscular atrophy, with a peculiar partial anæsthesia, trophic disturbances, and scoliosis.

Pathological Anatomy.—The seat of the disease is the substance of the cord. On opening it by transverse sections one finds one or more cavities extending in various degrees up and down. The most frequent primary site is the cervical swelling. From here it usually passes down and may reach the whole length of the cord. It also extends upward and may involve

the medulla and the nuclei of the cranial nerves. The cavities are of irregular shape, small size, and filled with a liquid like the cerebro-spinal fluid. They are situated oftenest posterior to the commissure and involve one or both posterior horns, but they may be so extensive as to involve almost the whole of the centre of the cord at some levels. The walls are usually lined by a membrane and surrounded by a gliomatous tissue (Figs. 130-135).

This membrane is composed of a rather dense gliomatous tissue. It may be absent in some parts. The *glia* cells are in various stages of development and degeneration. In parts of the cord the new growth may form a large and solid mass occupying

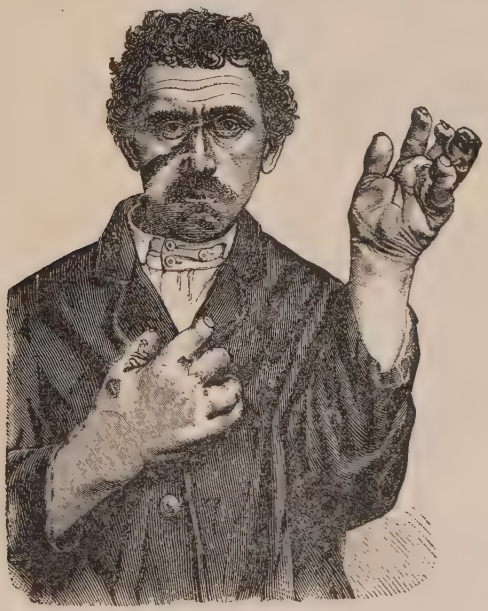


FIG. 129.—SHOWING HAND IN SYRINGO-MYELIA OF TYPE OF MORVAN'S DISEASE (ERB).

most of the centre of the cord. Small hemorrhages and foci of myelitis may be present. In some cases there is evidence of a dilated central canal, with neuroglia hyperplasia of the walls and a gliomatous infiltration about this. The epithelium of the central canal may form part of the wall of the cavity.

Pathology.—At about the sixth week of fœtal life the central canal of the cord is large, diamond-shaped, and reaches nearly to the anterior and posterior surfaces of the cord. This cavity gradually contracts and unites in the middle, the anterior part forming the central canal and the posterior part the posterior septum. The posterior part may, through some embryological defect, fail to close, and a congenital cavity may be left. About this a glioma may develop, and we have syringo-myelia. The central canal

may remain imperfectly contracted, and a syringo-myelia may develop in connection with it. Probably most cases of syringomyelia are developed on the basis of an embryological defect.

Gliomata were formerly thought to be practically identical with sarcomata. It is believed now, however, that they are of epiblastic origin, and like nerve cells are modifications of epithelial tissue. This tissue in its normal state is called neuroglia. It is a nervous substance. It is composed of small cells, round or of irregular shape, with a large nucleus and fine fibrillary prolongations. In glioma these cells are relatively much more numerous, while the fibrillary network is less conspicuous. There is considerable variation in the relative richness of cells and fibres, however. When the former are very frequent the term gliosarcoma has been wrongly given to the tumor. The glioma is penetrated by small blood-vessels whose walls are often diseased, so that minute hemorrhages occur and the glioma becomes stained and pigmented. In other cases it is gray or yellowish in color.

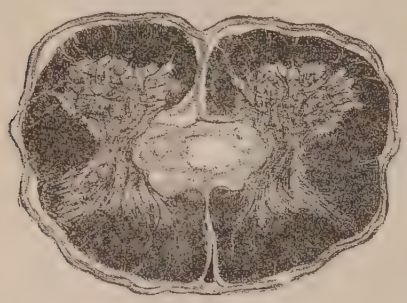


Fig. 130.

Fig. 131.

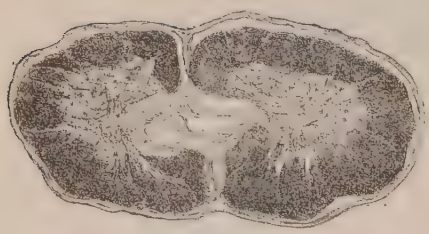


Fig. 132.

Figs. 130–132.—Sections of Spinal Cord at Different Levels in Syringo-Myelia (Bruhl).

The rich cellular proliferation in gliomata has suggested an analogy in its growth to that of inflammation, and the term gliosis is used as analogous for neuroglia to inflammation for connective tissue. Gliosis differs from sclerosis, however, in the fact that in the latter process the multiplication of fibres dominates, while in the former it is the cells; besides this, in gliosis there is a tendency to softening and formation of cavities; and all nerve fibres disappear. In sclerosis some nerve fibres remain, and one observes the presence of granular and amyloid bodies.

Peculiar Types.—1. The disease may be latent, giving rise to very few symptoms or to none that are characteristic.

2. There may be a period of irritation and pain in the extremities followed by paraplegia, with few sensory troubles, the course suggesting a chronic transverse myelitis or a Brown-Séquard paralysis.

3. There is a type in which bulbar symptoms develop early, but differing from ordinary bulbar paralysis in the involvement of the trigeminus and other cranial nerves not commonly attacked.

4. There is a form characterized by a rather rapid ascending paralysis.

None of these types can ordinarily be recognized during life.

5. There is a type characterized by the symptoms of muscular atrophy with analgesia and felons (Morvan's disease). In this type there is probably a complicating neuritis. Some assert that all cases of Morvan's disease are cases of syringomyelia, but this is not proven.

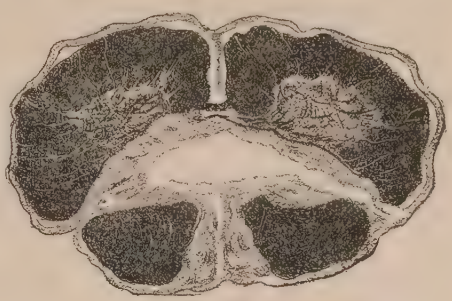


FIG. 133.

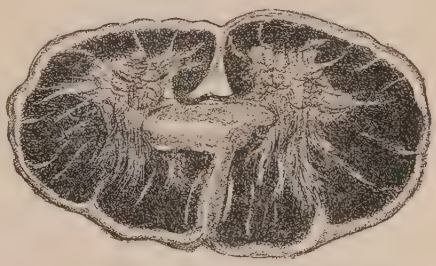


FIG. 134.

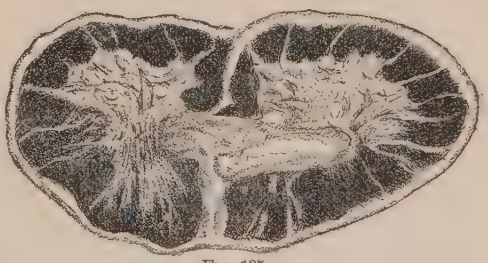


FIG. 135.

FIGS. 133–135.—SECTIONS OF SPINAL CORD AT DIFFERENT LEVELS IN SYRINGO-MYELIA (BRUHL).

Diagnosis.—The disease is distinguished in its classical form by (1) its beginning at the *period of adolescence*, (2) by the progressive muscular atrophy combined with the peculiar dissociated disturbances of sensibility, (3) by the trophic disturbances and scoliosis.

It has to be distinguished from progressive muscular atrophy and dystrophy, and amyotrophic lateral sclerosis, hypertrophic cervical pachymeningitis, chronic transverse myelitis, Morvan's disease and anæsthetic leprosy.

The sensory and trophic disorders and scoliosis enable one to distinguish it from progressive muscular atrophy. In leprosy

the dissociation of the sensory symptoms is not present, and the anæsthesia is distributed along the course of the nerves or in sharply circumscribed plaques. In some cases the peculiar tubercular disease of the skin and the history of the case make the diagnosis easy. In leprosy, also, there is a perineuritis, and the enlarged inflamed nerves may be felt. Portions of the skin may be excised and examined for the leprous bacillus. As regards the differentiation from Morvan's disease, this cannot often be done. Still whitlows are rare in ordinary forms of syringomyelia. Morvan's disease begins in one hand and slowly extends, with remissions, to the other. Usually there is loss of tactile as well as thermic and pain sense.

The prognosis so far as life is concerned is bad. But the disease has often a long course, ranging from five to twenty years, and periods occur in which the progress of the disease seems arrested and improvement occurs.

Treatment.—It is not impossible that we may find some drug which acts specifically on gliomatous tissue, checking its growth. At present we know of only two things which may possibly do this: nitrate of silver and arsenic. These drugs should be given; and for the rest, tonic and symptomatic treatment are indicated.

FUNCTIONAL DISORDERS.

Spinal Irritation.

This name is given to a functional disorder characterized by great pain and tenderness along the spine and associated with a neurasthenic and hysterical condition. The disease has been described as spinal hyperæmia (Hammond) and as spinal anæmia.

Etiology.—Spinal irritation is far more frequent in women than in men. It occurs between the ages of fifteen and thirty-five chiefly. A neuropathic constitution usually exists. The exciting causes are mental shocks and trauma, which latter need be only slight; excessive physical and mental work in young people; uterine displacements, sexual irritation, eye strain; and sometimes infectious fevers. I have seen cases which were the residuum of sporadic cerebro-spinal meningitis.

Symptoms.—The disease usually comes on slowly, with feelings of tenderness, pain, and burning in the back. Walking increases this; sitting and using the arms soon do the same. The neck tires on any exertion; and severe paroxysms of a boring neckache or of headache or backache occur. The patient has to lie down nearly all day, and finally in bad cases remains in bed. There are tender points on each side of the spine, generally at about the level of the sixth cervical, fourth to sixth dorsal, and dorso-lumbar junction. These points vary in position and sensitiveness. Pressure on them may cause nausea, pulse disturbances, paræsthesia, or radiating pain. The skin over the back is

sensitive, and there may be sensitive points over the ovaries and epigastrium.

The reflexes are exaggerated; there is some motor weakness, and the patient may be unable to walk, stand, or use the arms very much, but this is due more to the weariness and pain that result than to actual paralysis. Some wasting occurs in the limbs, but degenerative reactions are never found. Occasionally the patient has cramps and twitchings in the extremities. Sometimes there occurs in spinal irritation a nearly complete paraplegia. This is usually hysterical and always is functional. When such a combination of spinal irritation and paraplegia is caused by injury, it is called "concussion of the spine." Mental symptoms such as undue despondency, irritability, nervousness, and emotional crises are often present. Neither hysteria nor mental feebleness, however, necessarily characterizes spinal irritation. Many patients are unusually bright, hopeful, and intelligently anxious to recover. Sleep is usually poor and disturbed by dreams. The hands and feet are often cold and clammy; the pulse is rapid and of variable tension. The urine is of light specific gravity, with relative excess of phosphates.

Visceral symptoms are common in most cases. They consist of sensations of respiratory oppression, palpitations, anorexia, nausea and vomiting, constipation alternating with diarrhœa, attacks of polyuria, and sometimes vesical and genital weakness. The symptoms are usually exaggerated at the menstrual period.

The disease runs a chronic course; it always lasts months, often years, depending largely upon treatment and surroundings. Some of the symptoms of the trouble may remain for a lifetime.

Pathology.—The symptoms of the disease correspond exactly to what would follow from an irritation of the spinal meninges and posterior branches of the mixed spinal nerves. Hence the name spinal irritation can hardly be improved upon at present. The fact that classical cases follow an acute meningitis leads me to believe that a hyperæmia generally underlies the condition.

Diagnosis.—This must be made from cervical caries, organic disease of the spine and of the cord. In caries the rigidity, deformity, and localized pain are generally sufficient helps. Chronic meningitis is distinguished by the history of an acute onset, the symptoms are more stable, there is more rigidity, and the pains are extrinsic and radiating. The hysterical symptoms are absent, and the tendency, on rest, is always toward recovery. If there is a chronic meningo-myelitis or myelitis, the paralyses will be perhaps greater, and there may be degenerative muscular changes, anæsthesia, and involvement of the sphincters. In a functional palsy there cannot be complete degenerative electrical reactions or permanent sphincter paralyses.

Prognosis.—Nearly all cases get well eventually. Persons with a weak will, feeble body, and neuropathic constitution have the worst outlook. Traumatic cases, if uncomplicated, are not much worse than others.

Treatment.—The treatment must be a mental as well as physical one. The patient may be put to bed at first, but she should be kept employed and interested if possible, and she should soon be encouraged to walk and work a little. Her surroundings should be not too sympathetic, and her daily life should be scheduled exactly for her. She should be well fed, but not stuffed. Internally it is customary to give strychnine, iron, mineral acids, salicine, and quinine as tonics; moderate courses of bromides interrupted at times, or small doses of codeia and valerian, may be used as sedatives. Indian hemp and the analgesics may be of service. I have seen no good from arsenic, nitrate of silver or gold. Iodides and the salicylates, however, are useful. For sleeplessness give a draught of quassia water at night, and make the patient lie on a hop pillow or inhale the steam from boiling water in which hops have been thrown. Locally, counter-irritation along the back must be kept up at stated intervals. For this purpose one may use mustard, iodine, blisters, the cautery, and the galvanic current. The faradic current with a wire brush is most efficacious for removing the cutaneous hyperæsthesia (Anstie) if the patient can be persuaded to stand the pain. Cups, leeches, Baumgartism and splints, jackets and braces all may have to be resorted to eventually. A very great deal depends upon the perseverance and personality of the physician.

Spinal Exhaustion (Spinal Neurasthenia).

This is a condition which is nearly the opposite of spinal irritation. It occurs as often in men as women and during middle life or the climacteric.

Etiology.—It occurs oftenest in brain-workers and the educated classes. The principal exciting causes are shock, injury, overwork, sexual excesses, and exhausting diseases.

The *symptoms*, which come on slowly, consist chiefly of a general motor weakness and condition of exhaustion. The patient cannot walk far or stand long. His reflexes are weak, the muscles get wasted and flabby. There are some pains and burning or creeping feelings along the back when any exertion is made. The stomach is weak, the bowels irritable, the sexual power nearly gone. Headaches, insomnia, mental depression, and hypochondriasis are present. The disease, in fact, presents many of the spinal and neurasthenic characters of spinal irritation, but the pain is less and the motor weakness much greater.

When this latter reaches the point of paraplegia and follows shock or injury, the trouble is also called "concussion of the spine." Whether an anæmia underlies it we do not know. There is certainly a nutritional defect.

The diagnosis, course, and prognosis are much the same as in spinal irritation.

Treatment.—The disease may be associated with sprains or surgical injuries which need attention. Aside from this, the treatment must be largely in the nature of the rest cure. Massive doses of tinc. nux vomica may be used (℥ xxx. to xl. t. i. d.), combined sometimes with a little opium or codeia. At the same time, electricity, massage, baths, and strong feeding are required. The patients usually improve on first going under treatment, then relapse a little. The physician must keep something in reserve for these relapses.

CHAPTER XVI.

ANATOMY AND PHYSIOLOGY OF THE BRAIN.

Anatomy.—The nervous system is developed from a hollow tube formed by a folding of the epiblast. The brain or encephalon grows out from its anterior part. This swells into three cavities called the anterior, middle, and posterior cerebral vesicles. From the anterior a secondary vesicle develops; the posterior divides into two: so that eventually there are five vesicles. Out of them the different parts of the brain are formed.

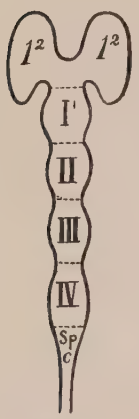


Fig. 136.

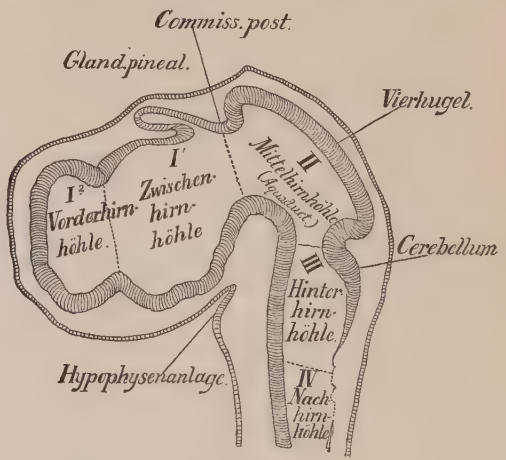


Fig. 137.

Fig. 136.—The Cerebral Vesicles.

Fig. 137.—Further Development of Vesicles into Fore-brain (Vorderhirn), 'Tween-brain (Zwischenhirn), Mid-brain (Mittelhirn), Hind-brain (Hinterhirn), and After-brain (Nachhirn). The cavities (Höhlen) of the vesicles are being formed into the ventricles (Edinger).

1. From the secondary anterior vesicle there grow the cerebral hemispheres, corpus callosum, fornix, anterior commissure, and corpus striatum. This forms the fore-brain.

2. From the primary anterior vesicle, the optic thalami, optic commissure, infundibulum, and corpora albicantia. This forms the 'tween-brain. The olfactory lobes and optic nerves are connected with this vesicle.

3. From the middle cerebral vesicle there grow the corpora quadrigemina and cerebral peduncles. This is the mid-brain.

4. From the fourth vesicle, the cerebellum and pons. This is the hind-brain.

5. From the fifth vesicle, the medulla, which constitutes the after-brain.

The development of these parts is shown in the accompanying diagrams. In man the fore-brain is enormously developed, the

FIG. 138.—STILL FURTHER DEVELOPMENT OF VESICLES. The cerebrum is being formed out of the first vesicle 1² (Edinger).

'tween-brain moderately developed, the olfactory lobes atrophic, the mid-brain almost rudimentary, the hind-brain well developed, the after-brain relatively not much developed. In the process of development of the brain, the neural canal becomes variously enlarged and constricted, until the ventricles of the brain, the foramina of Monroe, and the aqueduct of Sylvius are formed. The ventricles are the two lateral, the third, fourth, and fifth.

FIG. 139.—FURTHER DEVELOPMENT. The dotted lines show the original subdivision into vesicles (Edinger).

The foramina of Monroe connect the lateral and third ventricles; the aqueduct of Sylvius connects the third and fourth ventricles.

The different segments of the brain as thus enumerated are composed of deposits of nerve cells forming gray matter or ganglia, and strands of nerve fibres connecting these ganglia. Most of the general description of these parts must be gotten from treatises on anatomy. I propose, however, to enumerate and de-

scribe the various ganglionic deposits found in the brain; then show the way in which they are connected with each other and the spinal cord.

The sixteen ganglionic deposits of the brain are arranged in the different segments as shown here:

1. Cerebral cortex.
2. Corpus striatum. { Caudate nucleus. / Lenticular nucleus. } Fore-brain.
3. Olfactory lobes.
4. Optic thalamus.
5. Corpora geniculata.
6. Corpora mamil.
7. Luy's body. } 'Tween-brain.
8. Corpora quadrigemina.
9. Red nucleus.
10. Substantia nigra. } Mid-brain.
11. Cerebellar cortex.
12. Cerebellar nuclei.
13. Pons nuclei. } Hind-brain.
14. Olivary body.
15. Nodal nuclei.
16. Cranial nuclei. } After-brain.

The Cortex Cerebri and the Convolutions.—The gray matter of the surface of the brain is called the cortex cerebri, and it is by far the largest and most important deposit of nerve cells in the body. The cortex is from 2 to 4 mm. (one-twelfth to one-fifth of an inch) thick, and its total superficial area is 1,800 to 2,700 sq. ctm. The area of gray matter lying in the fissures is about twice that lying on the surface (Donaldson). The cerebral cortex is arranged in folds or convolutions (gyri) separated by fissures or sulci. These fissures divide the brain also into lobes. The fissures of the brain are divided into *primary* and *secondary*. The former are permanent, and present little change in size, location, or direction. The latter are variable in all these respects, and are often called sulci for purposes of distinction.

The primary fissures of the brain are:

The longitudinal.
The transverse or choroidal.
The fissure of Rolando or central.
The fissure of Sylvius.
The parietal.
The parieto-occipital.
The calcarine.

The position of these fissures is indicated on the accompanying diagrams, which are based on descriptions of Wernicke, Eberstaller, Wilder, Cunningham, and many observations of my own. I have not space to give detailed descriptions.

The secondary fissures or sulci will be enumerated in describing the lobes. They are divided into the typical or more or less constant and the atypical or tertiary fissures.

The primary fissures divide the cerebrum into various portions called lobes. The lobes are:

Frontal.
Parietal.
Temporal.
Occipital.
The central or island of Reil.
Olfactory.
Limbic.

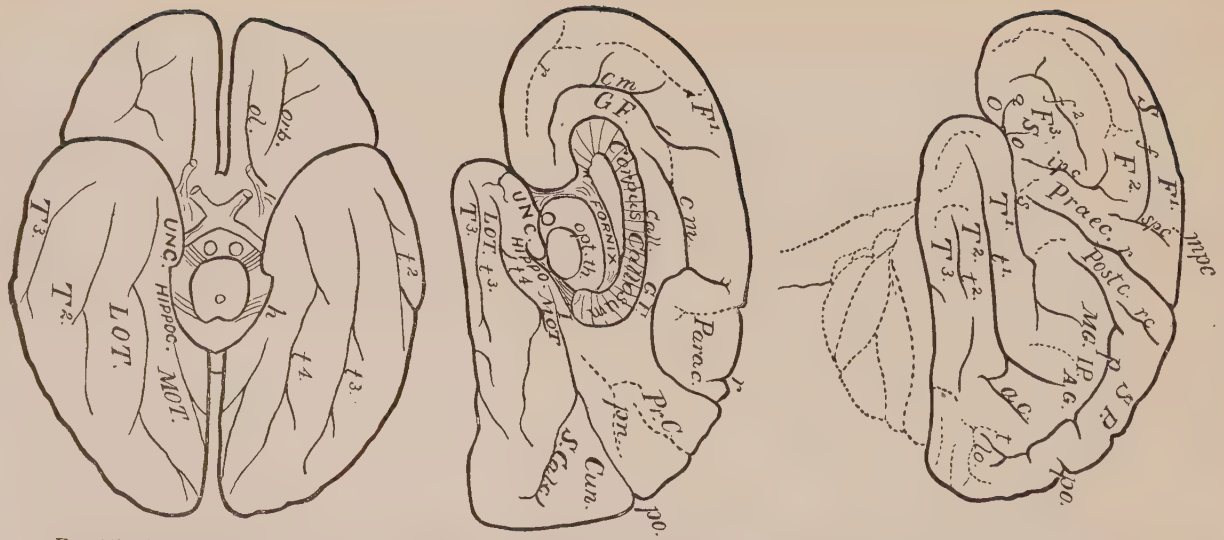


FIG. 140.- SHOWING CONVOLUTIONS AND FISSURES. (*Convolutions* in capitals.) F¹, First frontal; F², second frontal; F³, third frontal; O, operculum; *Præc*, Præcentral; *Poste*, post-central; S P, superior parietal lobule; M G, marginal gyrus, A G, angular gyrus, making the inferior parietal lobule; T¹, first temporal; T², second temporal; T³, third temporal; L O T, lateral occipito-temporal or lingual; M O T, median occipito-temporal or fusiform, which unites with UNC, uncinate, and H I P P O, hippocampal; *Cun*, cuneus; *PrC*, præcuneus; *Parac*, para-central; G P, gyrus fornicatus; I, isthmus; G F + I + *Hippo* + *Unc* = limbic lobe. (*Fissures* in Italics.) PRIMARY: S, Sylvian (*a*, anterior; *v*, vertical branch); *r*, Rolandic; *po*, parieto-occipital; *p*, parietal (inter-parietal); *call*, arcuate or callosal; *calc*, calcarine. SECONDARY: f^1, f^2, f^3, first, second, and third frontal; *r*, rostral (fronto-marginal); *orb*, orbital; *ol*, olfactory; *cm*, calloso-marginal (super-callosal, subfrontal); *mpc*, median præcentral; *spc*, superior præcentral; *ipc*, inferior præcentral; *rc*, retrocentral; *pn*, præcuneal; t^1, t^2, t^3, t^4, first, second, third, and fourth temporal; *h*, hippocampal; *lo*, lateral occipital; *ac*, anterior occipital (ascending temporal); *t*, transverse occipital (anterior occipital).

There are eight fissures and seven lobes.

The frontal lobe is limited behind by the fissure of Rolando, and on the median surface by the calloso-marginal or subfrontal fissure and its ascending branch. It has the following sulci: 1. Superior frontal and superior præcentral. 2. Inferior frontal and inferior præcentral. 3. Olfactory. 4. Tri-radiate.

In the frontal lobe are the following convolutions: 1. Præcentral or ascending frontal, having on its median surface (*a*) the paracentral lobule. 2. First or superior frontal. 3. Second or middle frontal. 4. Third or inferior frontal (Fig. 140).

The parietal lobe is limited behind by the parieto-occipital and anterior occipital fissures, below on its outer surface by the Sylvian fissure and a line drawn back from its upper end to the lower part of the anterior occipital. In front it is limited by the fissure of Rolando. On its median surface it is limited in front by the ascending branch of the calloso-marginal or subfrontal fissure, behind by the parieto-occipital, and below by the subparietal fissure. Its important sulci are: 1. The parietal or interparietal. 2. The post-central. The convolutions formed are, on the convex surface: 1. The post-central or ascending parietal. 2. The superior parietal lobule, having on its median surface (*a*) the quadrate lobule or præcuneus. 3. The inferior parietal lobule, which is composed of (*a*) the supra-marginal gyrus, (*b*) the angular gyrus. These latter gyri are variously described, but it is best to regard one, (*a*) the supra-marginal, as that surrounding the posterior end of the fissure of Sylvius; the other, (*b*) the angular, as that beginning at the upper limit of the first temporal fissure and extending back as far as the anterior occipital fissure.

The Occipital Lobe.—This is limited on its convex surface, anteriorly, by the parieto-occipital fissure, laterally to this by the anterior occipital and inferior occipital fissures. These in apes form a single continuous fissure sharply separating the occipital from the other lobes.* On its median surface the parieto-occipital fissure limits the lobe in front. On the under surface the anterior edge of the tentorium about marks the anterior limit.

The sulci are: 1. The transverse occipital (ape fissure of some writers). 2. The superior or lateral occipital. 3. The inferior occipital. On the median surface: 4. The calcarine, which joins the parieto-occipital. 5. The inferior occipito-temporal or fourth temporal.

The convolutions on the convex surface are: 1. The superior occipital. 2. The middle occipital. 3. The inferior occipital. 4. The descending occipital. On the median surface we find: 5. The cuneus. 6. Descending occipital.

The Temporal Lobe.—The convex or lateral surface shows the following sulci: 1. The first temporal or parallel sulcus. 2. The second or middle sulcus. On the under and median surfaces are: 3. The third or inferior temporal sulcus. 4. The fourth temporal or inferior occipito-temporal or collateral sulcus, which extends into the occipital lobe. 5. The hippocampal sulcus.

The convolutions are: 1. The first temporal convolution. 2. The second temporal convolution. 3. The third temporal convolution. 4. The lateral occipito-temporal or fusiform convolution. 5. The median occipito-temporal or lingual convolution. 6. The hippocampal convolution, which is an extension of

* There is still some doubt about the homology of these tissues.

4 and 5. 7. The uncinate convolution, which is an extension of 6.

The Island of Reil (lobus centralis, lobus caudicis).—This has a circular sulcus surrounding it, and several more or less short sulci dividing it into from five to seven short gyri.

The *limbic lobe*, if described as a separate lobe, is made to include anteriorly the gyrus fornicatus or gyrus cinguli, back of this the isthmus, then the hippocampal, uncinate, and dentate convolutions (Fig. 140).

The olfactory lobe is rudimentary in man. Its position is shown in the diagrams.

The operculum is the part of the brain that overlaps the island of Reil. It consists of a fronto-parietal part, formed by the lower end

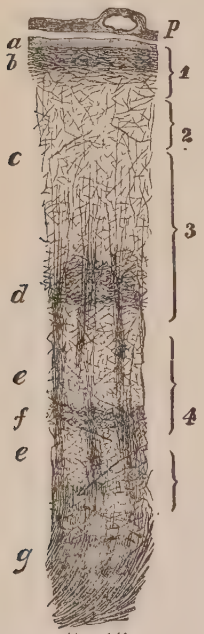


Fig. 141.

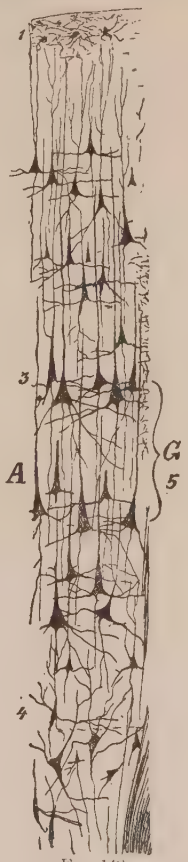


Fig. 142.

Fig. 141.—Section of Cortex of Frontal Lobe Stained by Weigert's Method, showing the fibre systems and layers. 1, 2, 3, 4, The ganglionic cell layers; *a*, neuroglia layer; *b*, tangential fibres; *c*, super-radial network; *d*, outer stripe of Baillarger or Glennari's stripe; *e*, inter-radial network; *f*, inner stripe of Baillarger; *g*, radial fibres of white matter (after Obersteiner and Edinger).

Fig. 142.—Section of Cortex of Frontal Lobe (after Golgi). 1, Neuroglia or barren layer; 2, small pyramidal layer; 3, large pyramidal layer; 4, irregular and spindle-cell layer. At G^5, a deposit of granular cells forms a fifth layer in the sensory occipital area of the cortex.

of the two central convolutions, a frontal part formed by the base of the inferior frontal convolution, and a temporal part formed by the tip of the temporal lobe.

The cuneus, præcuneus and paracentral lobule are important subdivisions on the median surface of the brain. Their position and boundaries are shown in the cuts.

Microscopical Anatomy of the Convolutions.—The cortex of the cerebrum is composed of nerve cells, a network of nerve fibres and processes, and of neuroglia tissue. Superimposed upon it is a very vascular membrane, the pia mater, which sends a rich plexus of vessels into it. We shall proceed to study: 1st, the structure and arrangement of the nerve and neuroglia cells; 2d, the arrangement and connections of the nerve plexuses. On both these points new facts are being constantly added, and the present description must be in many respects only provisional. 1st. The cells are arranged to a certain extent in layers. In the outer layer, next the pia mater, is a deposit of neuroglia tissue containing "spider cells." Beneath these are small, somewhat irregularly shaped pyramidal cells (angular cells of Lewis); next come large pyramidal cells; and deepest of all irregularly shaped cells (including the granule cells of Lewis) and spindle-shaped cells.

In the above I have described four layers of cells, and this may be considered the type. Some anatomists describe five typical layers, the fifth being made by a subdivision of the fourth (by Meynert) or of the third (by Lewis). The common four-layer type is found in the central convolutions and frontal lobe. In the occipital region there are six (Lewis) or eight (Meynert) layers described. These are formed by the interposition of granule cells which subdivide the third layer. Various types of cortex are described depending upon the different degree of development of the cell layers and upon the fibre arrangements. The common or motor type, as has been stated, has four layers. The large pyramidal cells are here numerous and are arranged in clusters. The sensory type has at least five layers, as described for the occipital cortex, and here the large pyramidal cells are few and isolated. The pyramidal cells are arranged with their apices pointing to the periphery. They give off apical, lateral, and basal processes. The basal process of the pyramidal cells is continued as an axis cylinder. Some pass down into the white matter: others turn up and enter the fibre systems of the cortex. The spindle cells point toward the periphery also, except at the bottom of the sulci, where they lie parallel to the surface. Their processes connect neighboring areas and are called associative fibres.

The small pyramidal and spindle cells measure about 10 x 18 μ; the large, 20 x 40 μ ($\frac{1}{1200}$ x $\frac{1}{600}$ in.). There are in the upper central and paracentral convolutions giant cells (of Betz) which measure 125 x 55 μ ($\frac{1}{200}$ x $\frac{1}{500}$ in.).

Despite the great variety and complexity of the cortex, it is probable that there are but three principal classes of cells: (1) those which receive nervous impulses and which lie chiefly in the second and granule layers; (2) those which associate and co-ordinate these impulses, and which lie in the second and deep in the fourth layers (small pyramidal and spindle cells); and (3) those which discharge impulses and which lie in the third layer (large pyramidal cells).

The fibres and plexuses of the cortex are composed of processes from the nerve cells and terminals coming in from the white matter. They collect into several close networks. One, lying just under the neuroglia layer and running parallel to the surface, is called the *tangential* layer of fibres; a second layer runs among the large pyramidal cells, forming the outer *stripe of Baillarger;* and a third layer, beneath this, is called the inner stripe of Baillarger. Besides these, there are radiating fibres, running in from the white matter and forming inter-radial and supra-radial networks (Fig. 142).

The cortical gray matter, as will thus be seen, contains layers of nerve cells, into which nerve fibres penetrate. These terminate, as do all fibres, in end-brushes, which surround the receptive

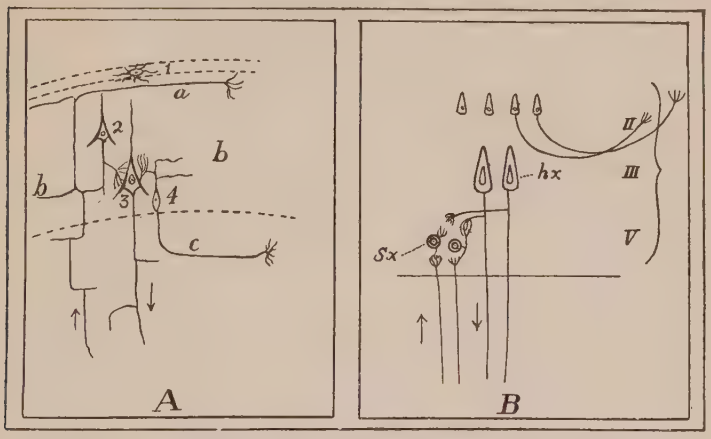


FIG. 143.—DIAGRAMATIC REPRESENTATION OF THE RELATION OF THE CELLS OF THE CORTEX TO EACH OTHER AND TO DISTANT PARTS. A, Motor type of cortex. 1, Neuroglia cell; 2, small pyramidal and granule cell; 3, large pyramidal cell; 4, spindle cell; *a*, tangential fibres; *b*, stripes of Baillarger; *c*, associative fibres. B, Sensory type of cortex (Von Monakoff). *II.*, *III.*, *V.*, Second, third, and fifth layers of cortex; *hx*, large pyramidal cell; *sx*, granule cells. In A, impulses are received by the small cells 2, distributed and associated by the tangential and other fibres *a*, *b*, *c* and by the spindle cells 4; impulses are emitted by the large cells 3.

or sensory cells. An enormous number of fine fibres are given off by the cells; some of these form layers in the cortex and connect neighboring parts, others run out and connect more distant parts or pass down to lower levels. There must be three kinds of fibres—afferent, associative, and efferent—just as there are three types of cells (Fig. 143).

The different convolutions and lobes of the brain are connected with each other by association fibres and with parts below by projection fibres.

The *association* fibres consist of short fibres connecting neighboring convolutions, long fibres connecting neighboring or distant lobes, and commissural fibres connecting the two halves of the cerebrum.

The short association fibres are numerous; they lie close beneath the gray matter and connect convolutions of the same lobes. The course of many of the long association paths is not yet well known, the descriptions commonly given being not altogether correct (H. Sachs). The occipital lobe is connected by long tracts to the temporal lobe, and perhaps slightly to the inferior parietal lobe (H. Sachs). The temporal lobes are connected only to the occipital. The frontal lobe is apparently connected with the parietal. Its connection with the temporal is denied (H. Sachs). All the lobes of the two halves of the brain are connected with each other by commissural tracts in the corpus callosum and anterior commissures.

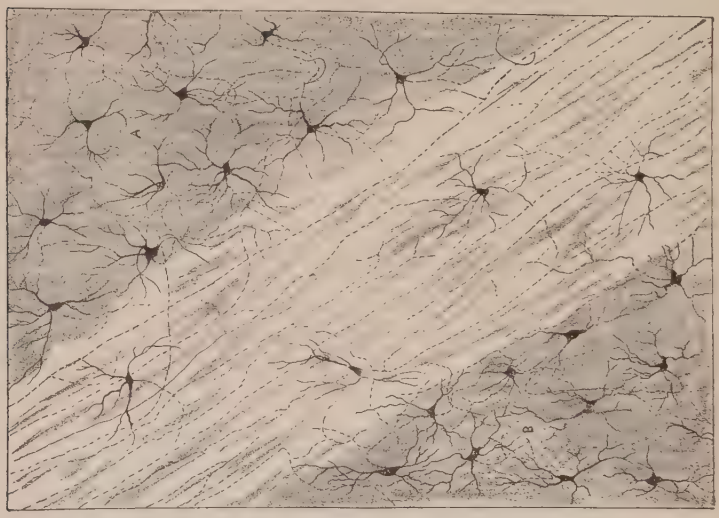


Fig. 144.—Nerve Cells of Corpus Striatum with Processes Running into Internal Capsule.

I will describe the projection fibres later.

The corpora striata are composed of two parts: the caudate nucleus and lenticular nucleus. These two nuclei are separated dorsally by the white fibres forming the internal capsule. Below they are continuous. Their shape and relations are not easily appreciated except by actual inspection of specimens. They are indicated in the accompanying figures (148–150). The head of the caudate nucleus is connected with the gray matter of the anterior perforated space. The tail extends into the temporal lobe, where it is continuous with the gray matter of the cortex, called at this point the amygdalum, and with the claustrum.

The lenticular nucleus is also continuous with the gray matter of the anterior perforated space. It is divided into three portions; the external is called the putamen, the two inner the globus pallidus. These parts are separated by white fibres. The caudate nucleus, putamen, and probably all of the corpus striatum are modifications of the cerebral cortex.

The corpus striatum has (1) fibres which pass to it from the cortex, (2) fibres which pass through it from the frontal and parietal cortex, (3) fibres which originate in it, and (4) fibres connecting its different parts.

1. The fibres joining the ganglion to the cortex are few and merely associative.

2. The fibres which pass through it pass chiefly into the dorsal part of the cerebral peduncles (or tegmentum) and are connected with the sensory tracts there.

3. Fibres which originate in the caudate nucleus and putamen collect together and join with the cortical fibres to form the

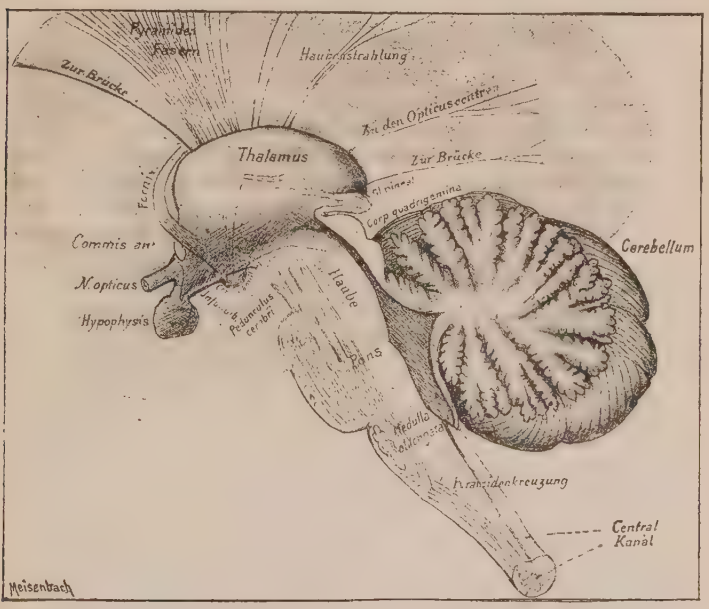


FIG. 145.—SHOWING THE RELATIONS OF THE OPTIC THALAMUS TO OTHER PARTS (EDINGER).

"lenticular loop" (ansa lenticularis). Some go to the subthalamus (Luy's body) and optic thalamus; most go to the inferior olives and thence connect with the cerebellum. Some perhaps go to the posterior longitudinal bundle, which is a band of commissural fibres that connect together the cranial nerve nuclei in the medulla.

4. Fibres connect the caudate nucleus and putamen with the globus pallidus.

The corpora striata are relatively rudimentary ganglia in man. They contain sparsely distributed multipolar and fusiform cells, $\frac{1}{1\,0\,0\,0}$ to $\frac{1}{5\,0\,0\,0}$ in. in diameter, the larger being in the lenticular nucleus. They are chiefly of the sensory type (Marchi—Fig. 144).

The Optic Thalami.—These ganglia form the chief part of the 'tween-brain. They lie at the base of the brain, posterior and in-

ternal to the corpora striata. They are continuous with each other by means of the middle gray commissure. The upper or dorsal surface forms part of the wall of the lateral ventricles; the mesial surface forms the lateral wall of the third ventricle. Externally is the band of white fibres called the internal capsule; below this is a rather large mass, the red nucleus, external to a small gray nucleus called the subthalamic ganglion (Luy's body). Around and below these is a complex network of fibres called the stratum intermedium. Below this, on the base of the brain, are the corpora mamillaria. The thalamus is composed of four nuclei —the anterior, lateral, and median or posterior. The last is called the *pulvinar*. Besides these there is on the median surface a small ganglion called the g. habenulæ. The optic tracts wind around the posterior and outer edge of the thalamus; and connected with these and the thalamus are two other ganglia, the

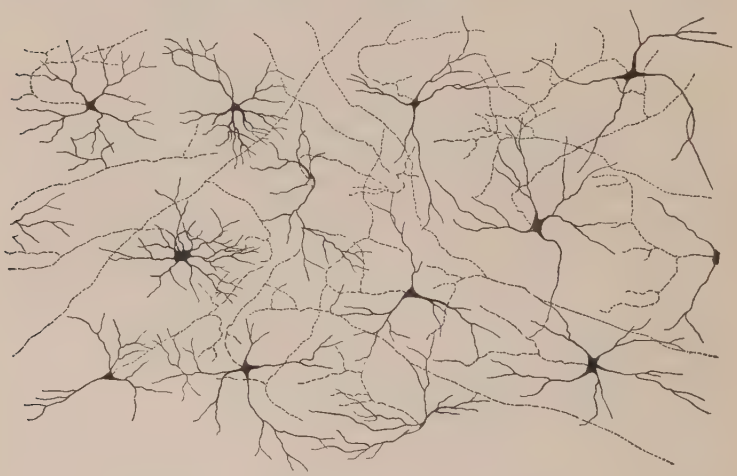


Fig. 146.—Nerve Cells of Optic Thalamus (Marchi).

external or lateral and internal or median *geniculate bodies*. Thus the thalamus is in close anatomical relations with five small ganglia; three below it—the corpus mamillare, red nucleus, and subthalamus; two postero-external to it, the geniculate bodies. To these may be added the corpora quadrigemina. The thalamus is composed of multipolar nerve cells rather more numerous and larger than in the striatum, but not grouped closely together. Nerve fibres pass in among these cells, which are said (Marchi) to be of the motor type (Figs. 145, 146).

The Corpora Quadrigemina.—The mid-brain contains as its chief ganglia the corpora quadrigemina. These consist of four tubercles, two anterior and two posterior (Fig. 145). In man they are rudimentary in structure and relatively unimportant in function. In man also the posterior tubercles are developed more relatively than in most lower animals. They together measure about 14 mm. ($\frac{1}{2}$ in.) in sagittal direction. In front lies the pineal gland and third ventricle; beneath is the aqueduct of Sylvius and the

structures of the upper pons and cerebral peduncles. This inferior boundary is shown by the dotted line in the cut (Fig. 147). Micro-

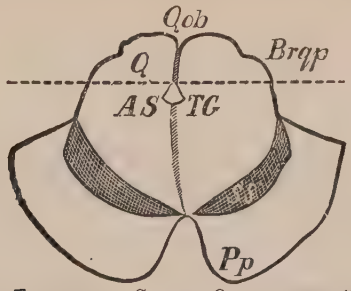


FIG. 147.—SECTION THROUGH THE CORPORA QUADRIGEMINA (Q), TEGMENTUM (TG), AND CEREBRAL PEDUNCLES. SS, Substantia nigra; *Pp*, peduncles; *AS*, aqueduct of Sylvius.

scopically the anterior lobes consist of layers suggesting a cortical type (Spitzka); the outermost is made up of optic-tract fibres, the next is a thin layer of small nerve cells, then optic fibres again,

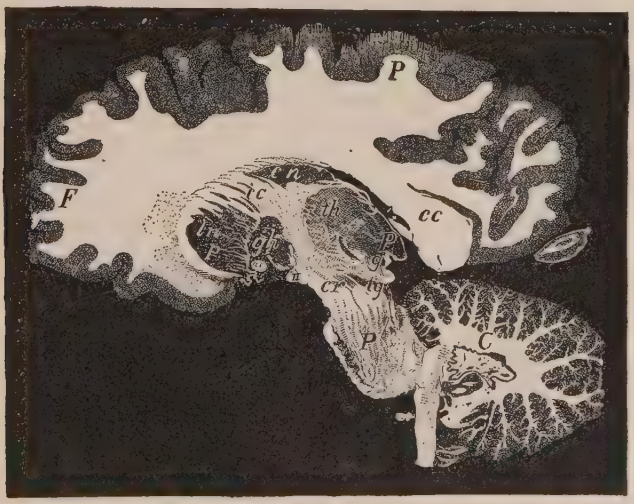


FIG. 148.—LONGITUDINAL SECTION, showing the lenticular (*ln*) and caudate nuclei (*cn*) separated by the internal capsule (*ic*), the corpus callosum (*cc*), the anterior commissure (*ac*), the optic tract (II.), the optic thalamus (*th*), the corpora geniculata (*g*), and the pulvinar (*p*), the crusta (*cr*), the tegmentum (*tg*), pons (P), and cerebellum (C); *p*, putamen; *g*, globus pallodus (G. Stewart).

and deepest of all a layer containing a few large cells. The posterior lobes are more homogeneous and contain small multipolar cells and a ganglion.

The accompanying figures show the relationships of the basal ganglia and other parts.

The Substantia Nigra.—Below the corpora quadrigemina and lying between the upper, sensory part (*tegmentum*) of the peduncles of the brain and the lower motor part (*crusta*) is the substantia nigra. It contains large multipolar, angular, and fusiform cells deeply pigmented (Fig. 147).

The Red Nucleus.—Dorsal to the s. nigra and at about its

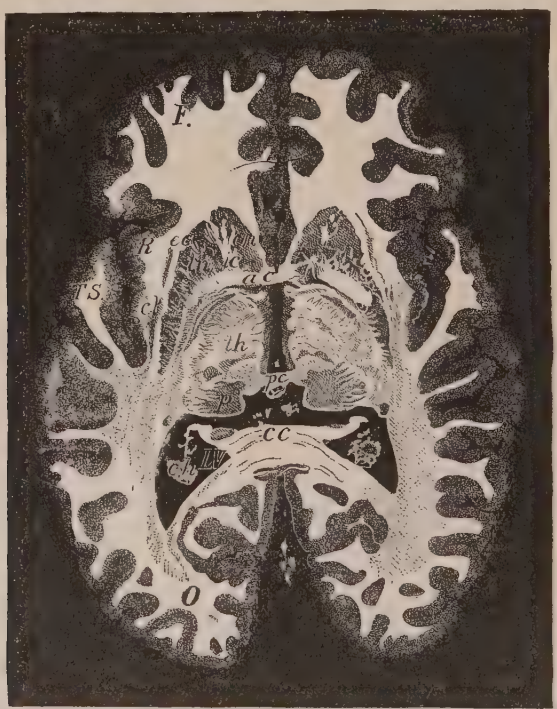


Fig. 149.—Horizontal Section, showing the frontal (F), temporo-sphenoidal (TS), and occipital lobes (O), with their gray and white matter, the island of Reil (R), its gray and white substance, the claustrum (*cl*), the external capsule (*ec*), the lenticular nucleus (*ln*), the caudate nucleus (*cn*), the internal capsule (*ic*), the optic thalamus (*th*), and pulvinar (*p*), the corpus callosum (*cc*), the anterior and posterior commissures (*ac* and *pc*), the lateral ventricle (LV), with the choroid plexus (*ch*) (G. Stewart).

middle extent is the red nucleus. It is spherical or oblong, very vascular, and contains numerous small cells.

The subthalamus (Luy's body) lies more dorsally, but in about the same plane as the substantia nigra. It measures about $\frac{1}{8}$ by $\frac{1}{2}$ inch and contains a few cells and a very fine plexus of nerve fibres.

The position and relation of these bodies are shown in Figs. 156–158.

The nuclei of the pons varolii are irregularly distributed

masses of nerve cells lying deep among the longitudinal and transverse fibres. A special nucleus lying low down in the pons is known as the *superior olive*. In the *after-brain or medulla* we have the gray matter of the floor of the fourth ventricle and its cranial nerve nuclei, the olivary bodies, and certain small deposits of gray matter called the *nodal nuclei*.

The cerebellum in man consists of a median portion or vermis and two lateral hemispheres. It is connected to the cord and rest of the brain by anterior, middle, and posterior peduncles. It is composed of an external layer or cortex of gray matter, of white matter, and central ganglia. The gray matter lies in very close, narrow folds, producing with the white matter an appear-

Fig. 150.—Section Through the Middle of the Basal Ganglia, Præcentral (AF) and Part of Post-Central (AP) Convolutions, and (TS) Temporal Lobe. It shows the corpus callosum (*cc*), the fornix (*b*), the fifth ventricle (*v*), lateral ventricles (LV), small part of thalamus (*th*), internal capsule (*ic*), caudate nucleus (*cn*), lenticular nucleus (*ln*), external capsule (*ec*), claustrum (*ec*) (G. Stewart).

ance on section called the *arbor vitæ*. The vermis and hemispheres are divided by sulci into a number of lobes and lobules. The vermis is divided into superior and inferior portions. Its further subdivisions and those of the hemispheres are shown in the cuts (Figs. 151, 152).

In the white matter of either hemisphere is a nucleus of small multipolar cells, the corpus dentatus or ciliary body. To the median side of this, and belonging structurally to it, is a small nucleus, the emboliform nucleus. In the inferior vermis is a collection of larger multipolar cells, the *nucleus fastigium* or tegmental nucleus; just to the outer side, between it and the emboliform nucleus, is a small collection of cells, resembling those of the *n. fastigii*, called the nucleus globosus.

The cortex of the cerebellum is divided from within out into

a granular layer, a layer of large cells, and a molecular layer. The granular layer is composed of minute round cells and larger or Purkinje cells with processes. The cells are all of nervous character; possibly some belong to the neuroglia tissue. They

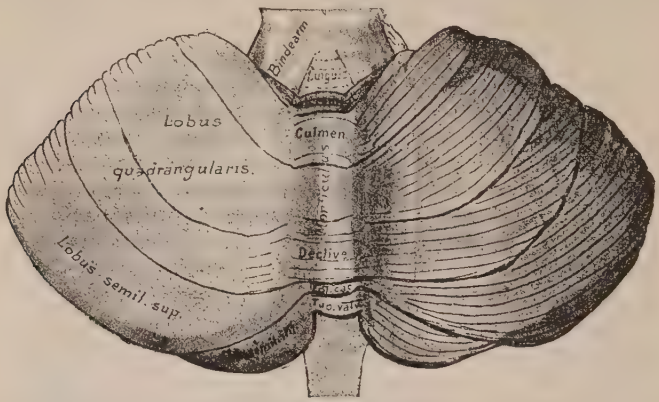


FIG. 151.—SHOWING THE LOBES AND OTHER SUBDIVISIONS OF THE CEREBELLUM, DORSAL SURFACE (EDINGER).

send up processes into the molecular layer, and some end in a network around the Purkinje cell. The large or Purkinje cells (20 x 30 μ) have very rich branching processes running up into the molecular layer as far as the periphery, where they turn down, and an axis-cylinder process running down into the white matter. The processes do not anastomose and the cells

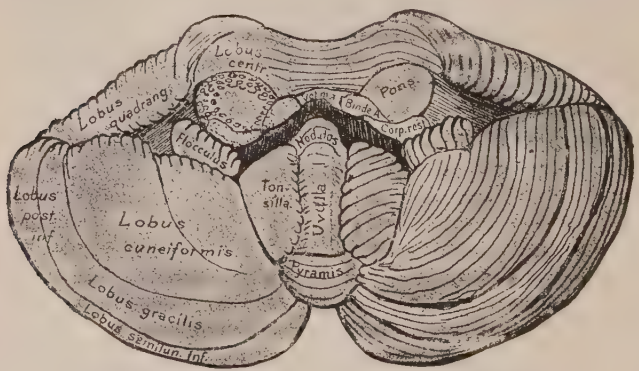


FIG. 152.—CEREBELLUM, VENTRAL SURFACE (EDINGER).

are not pigmented. The molecular layer contains large and small cells, some of which are multipolar, and send processes which end in a basket-work plexus around the Purkinje cells. Fibres coming in from the white matter appear to terminate also in end-brushes around these cells (Fig. 154—Cajal). The fibres of

the cortex are, as in the cerebrum, radiating and tangential. The tangential fibres are at the level of the Purkinje cells, among the processes of which they run. Just beneath the pia is a thin connective or neuroglia tissue membrane which sends radial fibres down through the gray matter, affording it a support.

It will be seen that the general arrangement of the cerebellar cortex is analogous to that of the cerebrum. Associative and receptive cells are found in the granular and molecular layers, and they send processes forming a rich network around Purkinje's cells which are efferent in function. The comparatively small number of the large cells is in harmony with the view that the cerebellum is an organ that receives and adjusts nerve impulses for co-ordinate distribution. All parts of the cerebellar cortex are anatomically alike.

Fig. 153.

Fig. 154.

Fig. 153.—Section Through Middle of Vermis and Pons.

Fig. 154.—Cerebellar Cortex of Embryo. D, Purkinje's cell; G, granule cell receiving a fibre and sending an end-brush or arborization to the P, Purkinje's cell (Cajal).

The white matter of the cerebellum consists of nerve fibres some of which go to form the peduncles. Others form anterior and posterior commissures running through the two extremities of the vermis and connecting the hemispheres. There is also a longitudinal commissure in the vermis. The white matter around the corpus dentatum is called the *fleece*.

Having described the general arrangement of the different divisions of the brain and the collections of gray matter found in them, we are prepared to study the tracts of white matter which connect the different parts.

The white matter, as already shown, is made up of:

1. Association fibres. { Short. Long. Commissural.
2. Projection fibres.

1. The association fibres of the cortex have been already described. Besides these, there are association fibres connecting different ganglia of lower levels. They are named Vicq d'Azyr's bundle, the fasciculus retroflexus or Meynert's bundle, the tegmental bundle, the brachia of the corpora quadrigemina and the posterior commissure. These may be called *ganglionic association tracts.* Some of these will be described later and indicated in the figures.

Commissural association fibres connect similar and symmetrical anatomical levels. They are the corpus callosum, the anterior

FIG. 155.—SECTIONS OF CORTEX OF SAME, BUT OLDER. A, Neuroglia layer; B, zone of bipolar cells; C, molecular layer; D, granular layer; *e*, *f*, *g*, bipolar cells of molecular layer; *j*, *o*, *y*, *n*, fibres from the white matter which end in end-brushes or arborizations capping the cells of Purkinje; *h*, Purkinje cell with axis cylinder and collateral.

and middle commissures, the fornix, and the anterior and posterior cerebellar commissures.

2. The *projection fibres* are long tracts which connect higher with lower levels. Some of them have a long and some an interrupted course. The long projection fibres extend from the frontal and parietal cortex down into the spinal cord. They pass as a narrow band of fibres between the lenticular nucleus and the caudate nucleus, helping to form the *internal capsule;* thence into the lower part of the cerebral peduncle, forming the crusta; thence through the pons and medulla. At the lower part of the medulla they form the anterior pyramids. About 90 per cent of the pyramids crosses over and passes down in the lateral columns as the crossed pyramidal tract; the other ten are continued down in

the anterior column as the direct pyramidal tract. Its fibres cross over in the anterior commissure. Both tracts connect with the cells of the anterior horns. The entire traject of fibres from the cortex cerebri to the anterior horns forms the *voluntary motor tract.*

The interrupted projection fibres consist of the sensory tracts and the cerebro-pontine tracts. The sensory tracts starting from the brain cortex in part connect with the thalamus, in part pass directly through the posterior portion of the internal capsule, then through the upper part of the cerebral peduncles, forming there the tegmentum, and thence through the pons and medulla as the lemniscus and formatio reticularis. In the medulla some fibres cross over, forming a "sensory decussation," and end in the nuclei of the columns of Goll and Burdach; others run in the formatio reticularis, pass down in the posterior and lateral columns, cross

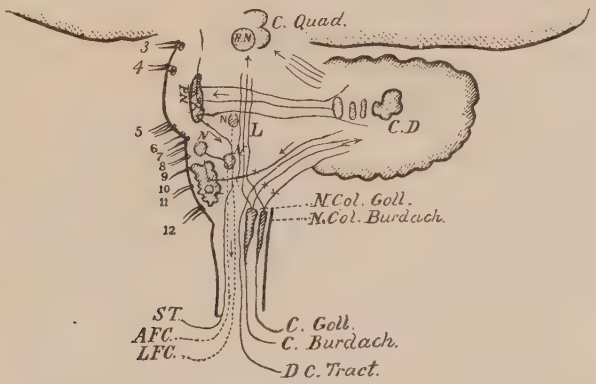


FIG. 156.—A, Connections of spinal cord with pons and medulla; N, nodal nuclei; O, olivary body; PN, pons nuclei; L, lemniscus; 3 to 12, cranial nuclei; *x*, decussation; ST, Sensory tracts; AFC, anterior fundamental columns; LFC, lateral fund. cols.; DC, direct cerebellar tract.

over at various levels, and connect with cells of the posterior horns. The cerebro-pontine tracts are described under Fig. 158.

Architecture of the Brain.—The relations of the ganglionic deposits of the brain with the cord and with each other cannot be described in any brief way. But by the aid of the accompanying diagrams the student can, after some study of ordinary descriptive anatomy, get an idea of the architecture of these parts. It is best to begin from below with the spinal cord, and show first the relations of its different columns to the medulla and pons varolii.

In Fig. 156 it will be seen that only the anterior and lateral fundamental columns of the cord are connected with the gray matter of the medulla and pons. The motor and sensory tracts pass through it uninterrupted. The antero-lateral fundamental columns connect with various deposits of gray matter in the medulla and pons which are called nodal nuclei. These in turn are connected with the cerebellum and pyramidal and sensory tracts. There is a long band of fibres in the medulla and pons called the

posterior longitudinal bundle. It is an analogue of the anterior fundamental column of the cord and connects the cranial nuclei. It is connected above with the basal ganglia.

Fig. 157 shows the relations of the spinal cord to the cerebrum and cerebellum. The pyramidal tracts, having mostly crossed in the lower medulla, go straight up through the internal capsule to the motor area of the cortex.

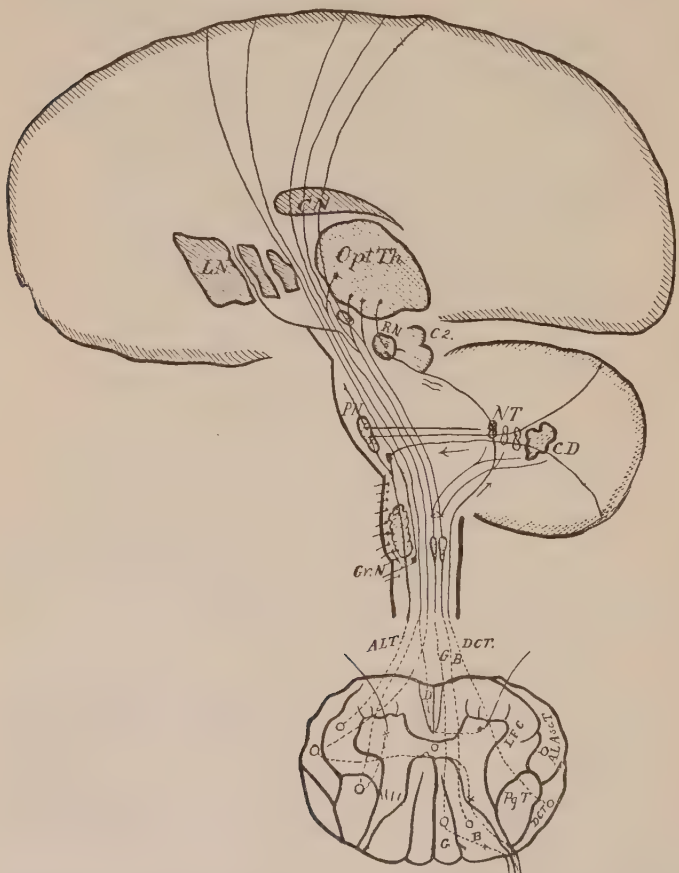


FIG. 157.—CONNECTIONS OF SPINAL CORD WITH CEREBRUM AND CEREBELLUM. Motor tracts go straight to cerebral cortex. Postero-internal and part of postero-external (sensory) tracts go to their nuclei, then to the cerebellum in part, to optic thalamus (and corpora quadrigemina ?) and lenticular nucleus and cerebral cortex. The antero-lateral ascending tracts go *via* formatio reticularis to optic thalamus and cortex. The direct cerebellar tract to vermis and then *via* anterior cerebral peduncle to opposite red nucleus and thalamus. The anterior fundamental and lateral fundamental columns to pons and nodal nuclei and to cerebellum

The sensory tracts cross in part, while part have crossed already in the cord. They go up, forming the inter-olivary tract, then the lemniscus or fillet. In part then they enter the dorsal portion or tegmentum of the cerebral peduncle, pass through the posterior part of the internal capsule to the motor area of the cortex and parietal lobe. Some fibres enter first the basal ganglia. The cord is connected with the cerebellum by the direct cerebellar tracts, the posterior columns, and antero-lateral fundamental columns. All these columns carry afferent impulses except those of the antero-lateral tracts, which are made up of associative fibres.

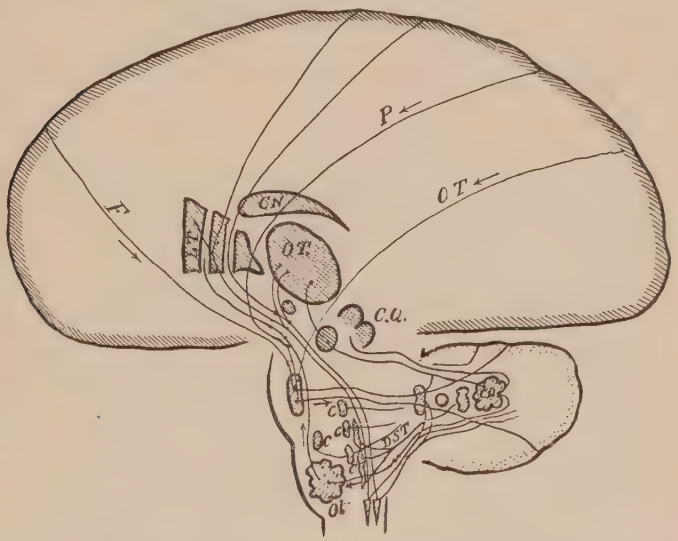


Fig. 158.—Connections of Medulla-Pons-Cerebellum with Higher Parts. Nuclei of columns of Goll and Burdach with optic thalamus, lenticular nuclei, and cerebral cortex. Olivary body, with lenticular nuclei and cortex. Pons nuclei with frontal and occipito-temporal cortex. Cerebellum, *via* middle peduncles, to pons nuclei and so to cerebrum. Cerebellum, *via* anterior cerebral peduncles, to red nucleus, optic thalamus (and corpora striata and brain cortex?), acusticus and trigeminus, posterior corpora quadrigemina.

In general, the spinal cord is connected with the vermis and fibres surrounding the dentate nuclei, the pons and medulla are connected with the cortex cerebelli and vermis, while the anterior peduncles arise chiefly from the dentate nucleus. (Marchi gives other relations.)

The connections of the pons, medulla, and cerebellum are shown in Fig. 158. The olivary body is connected with the cerebellum on the one hand and to the lenticular nucleus and cortex on the other. It is not connected with the cord. Impulses pass from the cerebellum to the olives, thence up to the brain. Nuclei in the pons are connected with the cortex of the cerebellum and with fibres running in the ventral part of the cerebral peduncle

on either side of the pyramidal tract, to the frontal and temporal and probably occipital lobes, and island of Reil. The nuclei of the acoustic, glosso-pharyngeal, and vagus, perhaps other sensory nerves, are connected with the cerebellum by a "direct sensory tract" which goes to the nuclei of the cerebellum. The nodal nuclei are connected, as has been stated, with the cerebellum.

Fig. 159 shows the relations of the optic thalamus and corpora quadrigemina to other parts. The corpora quadrigemina, by the posterior tubercles, are connected to the cerebellum and acoustic nerve, internal geniculate body and temporal lobes; by the anterior tubercles to the lemniscus and spinal cord, the

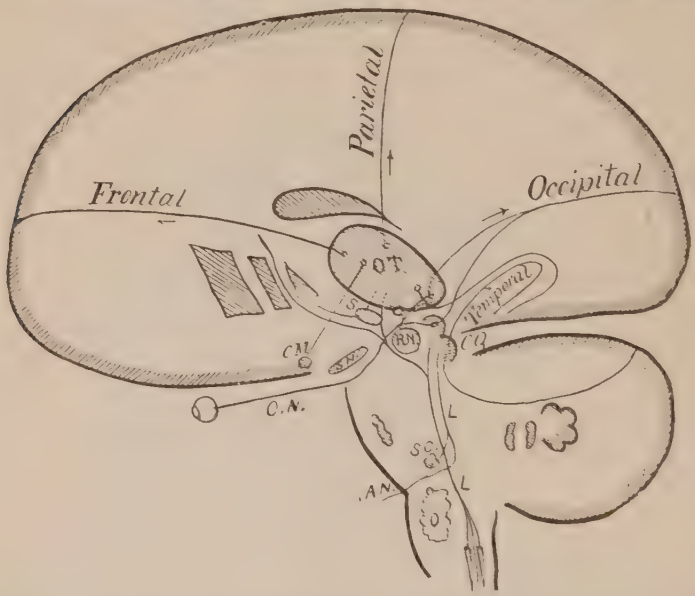


Fig. 159.—Relations of Corpora Quadrigemina and Optic Thalamus to Other Parts. OT, Optic thalamus; CQ, corpora quadrigemina; RN, red nucleus; S, Luy's body, or subthalamus; SN, substantia nigra; ON, optic nerve; AN, acoustic nerve; L, lemniscus; SO, superior olive.

optic nerve and optic thalamus, external geniculate body and occipital lobes. The thalamus is connected to the spinal cord by the lemniscus, to the medulla and cranial nerves by the posterior commissure, to the optic nerve and external geniculate body, to the cortex of the frontal, parietal, occipital, and temporal lobes, to the corpora mamillaria and fornix, and to the subthalamus and lenticular nucleus. All its connections cannot be shown or described; but it is the most richly connected ganglion of the brain.

Fig. 160 shows the relations of the corpus striatum to the other parts.

It is connected with the spinal cord by the lemniscus(?), with the red nucleus, subthalamus and thalamus, with the substantia

nigra, pons nuclei, and olives. Its connections with the brain cortex are slight and those with the motor tract doubtful. The corpus striatum seems to have its closest relations with the cerebellum, partly through the red nucleus, partly through the nuclei of the pons.

The Membranes of the Brain.—The membranes of the brain are the dura mater, the arachnoid, and pia mater. The dura mater lines the inner surface of the skull. It is attached loosely to the concavity, but closely to the base. It splits into two layers to form the venous sinuses of the skull. The inner of the two layers at certain points projects inward to form membranous

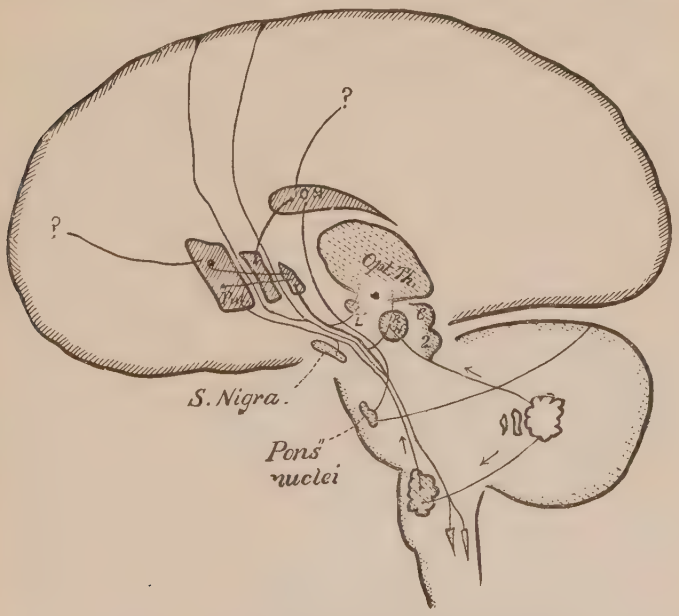


Fig. 160.—Relations of the Corpus Striatum to Other Parts.

septa. These are known as the great longitudinal or cerebral falx, the lesser longitudinal or cerebellar falx, and the tentorium. Hence both venous sinuses and membranous septa are formed out of the inner layer. The outer layer forms the periosteum of the bone. The dura mater is supplied with sensory nerves chiefly by the trigeminus, but posteriorly by the vagus. The blood supply will be described later.

The arachnoid is a thin, transparent, fibrous, non-vascular membrane lying between the pia and dura and continuous with the spinal arachnoid. It bridges over the fissures and the depressions at the base of the brain and forms between the pia and itself certain lacunæ or spaces. These are the central lacuna found at the beginning of the fissure of Sylvius, the callosal and those of the transverse fissures, and of the lateral aspect of the

pons varolii. The space between the dura and arachnoid is called the subdural or arachnoid cavity. It is lined with epithelium and resembles other serous cavities. The inner surface of the arachnoid is connected with the pia by numerous delicate fibrous processes. The space between these membranes is called the *subarachnoid space*. It communicates with the subdural space by means of the foramen of Magendie, which lies in the part of the arachnoid that passes over the pons and medulla, closing in the fourth ventricle. The subdural and subarachnoid spaces contain a serous fluid. The normal amount ranges from two drachms to two ounces, it being greater in old people. The arachnoid contains no nerves or blood-vessels. It is described by some as a part of the pia mater.

The pia mater lies beneath the arachnoid and is closely applied to the brain in all its folds. It is continuous with the spinal pia. It is very vascular and supplies the whole periphery and part of the interior of the brain with blood. It consists of two layers: an outer holding the larger vessels, and an inner delicate layer closely associated with the superficial neuroglia of the brain. The pia mater folds upon itself and passes through the transverse fissure into the third and lateral ventricles of the brain. These vascular folds form the velum interpositum, which gives off a choroid plexus to the lateral and third ventricles. Another fold, the inferior choroid plexus, is given off to the fourth ventricle. The pia mater has vasomotor, but no sensory nerves.

Functions of the Brain Membrane.—The dura mater, by its outer layer, acts as a periosteum; by its inner layer as a lymph sac. It is also, by virtue of its sensitiveness, a protection against injury and disease. The arachnoid forms the inner wall of the lymph sac. The pia mater is a vascular and nutritive organ. It is, however, also closely connected with the lymphatic system of the arachnoid. The blood supply and lymph supply of the brain vary in amount. In congestion the lymph can pass into the spinal canal or be rapidly taken up by the absorbents. In anæmia there may be compensatory increase of lymph. This fluid in disease may accumulate in the arachnoid sac, the subarachnoid space, or the ventricles, these spaces being all in communication with each other.

The Blood Supply of the Brain and its Membranes.—The vascular supply of the scalp, skull, and dura mater comes from the external carotids; that of the eye, brain, and pia mater from the internal carotids and vertebrals. The arrangement is shown here:

External carotid gives off:
- Occipital, inferior meningeal, arteries.
- Posterior auricular.
- Temporal:
  - Anterior.
  - Middle.
  - Posterior.
- Ascending pharyngeal, posterior meningeal.
- Internal maxillary, middle meningeal, small meningeal.

Internal carotid gives off:
- Anterior meningeal.
- Anterior cerebral.
- Middle cerebral.
- Posterior communicating.
- Anterior choroid.

Vertebral and basilar give off
- Posterior meningeal.
- Inferior cerebellar.
- Anterior cerebellar.
- Superior cerebellar.
- Posterior cerebral.

The general arrangement and distribution of the arteries of the scalp and dura is shown in the accompanying diagram (Fig. 161).

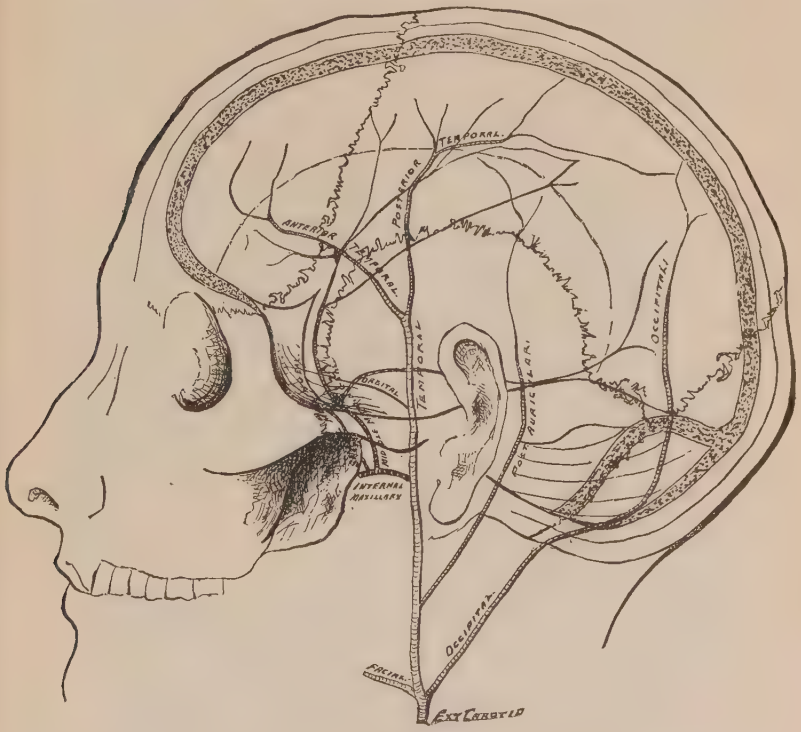


Fig. 161.—Showing the Blood Supply of the Scalp and that of the Dura Mater by the Middle Meningeal.

The *blood supply of the meninges* comes from the anterior middle and posterior meningeal arteries. These all come, except the small anterior meningeal branches and a small posterior branch, from the external carotid. The blood passes into the diploic veins, and from there passes chiefly into the lower occipital and lateral sinuses. Some of it, however, returns in the venæ comites. It all returns down toward the base of the skull. The most important of the arteries is the middle meningeal, both on account of its size and its distribution above important functional areas.

The *blood supply to the pia mater and brain substance* comes

from the internal carotid and the vertebral arteries. The branches of the former artery give off the anterior and middle cerebral, the posterior communicating, and anterior choroid. The vertebral arteries give off the inferior cerebellar, while the basilar branch of the vertebrals gives off the transverse, anterior cerebellar, superior cerebellar, and posterior cerebral arteries

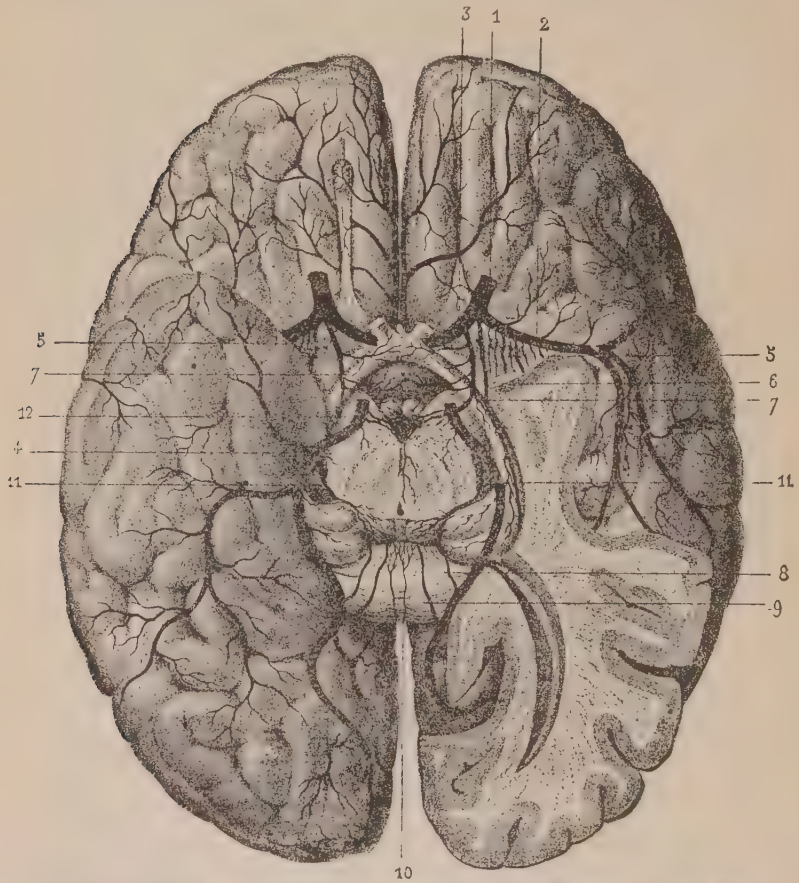


FIG. 162.—SHOWING THE ARTERIES AT THE BASE OF THE BRAIN. On the right side the brain is cut away, showing the cerebral arteries and the course of the posterior cerebral.

The cerebral arteries, anterior, middle, and posterior, are the three largest and most important. By their anastomoses the circle of Willis is formed. From the circle of Willis and the beginnings of the three arteries mentioned, several groups of vessels, six in all, are given off. They enter the base of the brain

and supply the great basal ganglia and adjacent white matter. They are called the *central arteries*, and they are the vessels usually affected in cerebral hemorrhages of adult life. They do not anastomose with each other. The *cortical arteries* are the terminal branches of the great cerebral arteries. They anastomose with each other but slightly. They are distributed very widely and carry much more blood than the central groups. Their distribution is shown in Fig. 163. The cortical arteries are distributed in the pia, and from there they pass in two sets, a superficial and a deep, into the gray matter, and for a short distance into the white matter. They pass straight in at right angles to the surface. They have richly arborescent branches which do not anastomose; consequently a knife plunged straight into the brain does not cut many vessels. The cortical arteries

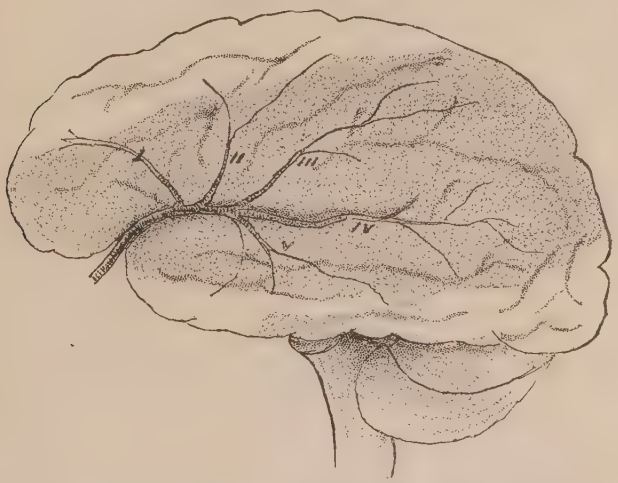


FIG. 163.—SHOWING THE DISTRIBUTION OF THE ARTERY OF THE SYLVIAN FISSURE, A PROLONGATION OF THE MIDDLE CEREBRAL. The area in front of the shaded part is supplied by the anterior cerebral, that behind by the posterior cerebral.

probably anastomose somewhat with each other, though not very freely. There is slight if any anastomosis between the cortical and central arteries. The pressure is thought to be less in the vessels of the gray matter.

The capillaries are surrounded by spaces called perivascular spaces which serve as lymphatic channels. The neuroglia cells send processes which connect with or form passages to the vessel walls (Fig. 164).

The blood of the convex and mesial cerebral surface, flowing up from the base, leaves the capillaries and enters veins. Thence it still passes upward, and for the most part enters the longitudinal sinus. The most of the vessels enter the posterior portion of the sinus and in a direction forward and upward, *i.e.*, against the current in the sinus. The course of the blood current is, therefore, opposed both to gravitation and to the venous flow.

The *veins of the cerebrum* are: 1, the superficial cerebral; 2, the deep cerebral veins; and, 3, the cerebral sinuses. The superficial cerebral veins are venæ comites. Those on the convex and mesial surfaces empty chiefly into the superior longitudinal sinus, as described; those on the basal surface empty into the cavernous and lateral sinuses. These veins have no valves, and their walls are very thin and without muscular fibres. The deep cerebral veins, or venæ Galeni, receive the blood from the lateral ventricles and from some of the central arteries supplying the basal ganglia. They empty into the straight sinus.

The *cerebral sinuses* are fifteen in number. The important ones are the superior and inferior longitudinal, the straight, the lateral, the occipital, the cavernous, and the superior and inferior

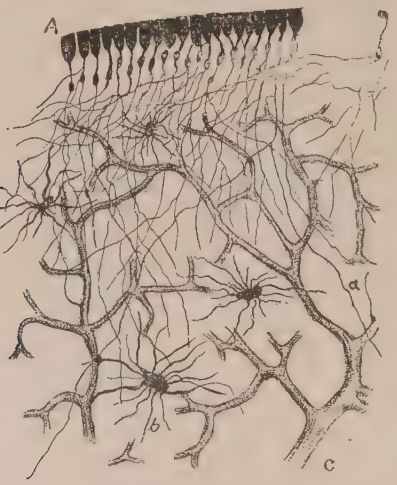


FIG. 164.—SHOWING THE NEUROGLIA CELLS OF THE BRAIN, THEIR RELATION TO THE BLOOD-VESSELS; ALSO THE SUSTENTACULAR PROCESSES OF THE EPITHELIAL CELLS OF THE LATERAL VENTRICLE (MARCHI). A, Epithelial cells lining lateral ventricle; *a*, process of same; *b*, spider or neuroglia cell; *c*, blood-vessel.

petrosal. They carry blood for the most part in a direction from before backward, and convey it eventually to the internal jugular.

Most of the blood of the convexity and mesial surface must pass into the longitudinal sinus, but there is a slight connection of some of the veins with the superior petrosal and straight sinuses. The superior longitudinal sinus also communicates slightly with veins of the scalp and with the facial vein. Some of the blood from the mesial surface also goes to the veins of Galen.

On the whole, however, the system of the convex and mesial cerebral surface is a close corporation, the blood having to pass into the superior longitudinal sinus and torcular Herophili, where it meets that of the straight and occipital sinuses, and flows forward through the lateral sinuses to the internal jugular. The circulation of the basal surface is less isolated. All the basal sinuses communicate with each other freely, and there are slight

communications between the veins of the scalp and the cavernous, lateral, and inferior petrosal sinuses. It is safe to tie any of the sinuses, except the lateral and the posterior part of the longitudinal. The cerebellar veins, superior, inferior, and lateral, empty into the straight, the lateral, and superior petrosal sinuses. None of the cerebral veins or sinuses have valves.

The pressure in the internal carotid arteries is about 150 mm., that in the cerebral sinuses 70 to 80 mm. (Gerhardt), and that in the jugular veins is almost negative. Both arteries and veins are more delicate than the extra-cerebral vessels.

Except its gray matter, the brain is not a very vascular organ, but this gray tissue ranks in richness of blood supply with the lungs and liver. The amount of blood in the brain at any one time is only about one to two per cent of the total blood in the circulation, or about four ounces (Ranke).

The diameter of the common carotids is 6.7 mm. (Thorne), that of the subclavians 6.2 mm., that of the internal carotids 4 mm., and that of the vertebrals 3.5 mm. (Gerhardt).* The blood to the brain, therefore, has passages three-fifths as great as the total arterial area near the heart, and the blood enters these passages at a pressure (250 to 150) three-fifths as great as that in the vessels near the heart.

The Functions of the Brain—Cerebral Localization.—The brain is the seat of conscious intelligence and mental activity. It has also control and direction of voluntary movements, it is the seat of instinctive acts, and it regulates in a measure the vasomotor, trophic, and secretory mechanisms of the body.

The Præfrontal Lobes.—The præfrontal lobes, or that part of the brain in front of the præcentral convolution, are concerned with volition and the power of self-control, concentration of thought and attention. The posterior part contains centres for the movements of the head and eyes. Injuries in this part of the brain produce changes of character, indicated by peevishness and irritability of temper, mental enfeeblement, lack of power to concentrate the mind or to control the acts or emotions.

The Central Convolutions, Paracentral and Upper Parietal Lobules.—This part of the brain is called the *motor area*, because it is concerned in the production of nervous impulses which cause voluntary motions of the body. Certain parts of this area are in relation with certain groups of voluntary muscles on the opposite side of the body. These areas preside not so much over single muscles as over those groups of muscles which act together in producing definite purposeful acts. The lower part of the central convolutions, known as the central operculum, is a centre for movements of the larynx, mouth, tongue, and face. Above this area and about the middle third of the central convolutions is the centre for the movements of the shoulder, arm, hand, and fingers. Still farther up, near the longitudinal fissure, and extend-

* J. Crichton Brown gives the last two diameters 2.8 and 2.2 mm. respectively.

ing over into the mesial surface and back into the superior parietal lobule, is the area for the trunk, hips, legs, feet, and toes. The base of the first and second frontal convolutions is the centre for movements of the head and eyes. The exact arrangement of these centres, which have been determined by experiments upon monkeys and other lower animals as well as by clinical and surgical observations on man, is shown in the accompanying diagrams. Probably the motor area is also the centre for the cutaneous sensations of the parts corresponding to the muscular groups which it supplies, so that what is called the motor is really a sensori-motor area. The motor area, when irritated by disease,

FIG. 165.—THE CORTICAL CENTRES.

produces paræsthesiæ and convulsive movements in the groups of muscles which it represents. Destruction of it causes not only a paralysis, but a certain amount of cutaneous anæsthesia.

The various sensori-motor centres are not sharply limited, but lap one over the other, so that the motor area for the forearm, for example, extends over somewhat into that for the shoulder. The corresponding sensory areas are still more diffuse, so that it takes a much more extensive destruction of a certain area of the motor cortex to produce an anæsthesia of the arm than it does to produce a paralysis of the arm (Figs. 165, 166).

Bilateral Representation.—Those muscles of the two sides of the body which act together have a double representation in the brain. For example, each group of muscles used in inspiration

has a centre in both hemispheres; consequently, when one centre is destroyed no paralysis results, for the reason that the other centre continues its work. In the same way some of the muscles of the face, such as those for closing the eyes, have a double representation, and a lesion destroying the centre for the orbicularis palpebrarum on one side will not usually cause paralysis, because of the continued action of the centre of the other side. The more perfect and habitual the associated action of the muscles of the two sides of the body, the more completely can one centre do the work of its associate. The best examples of the muscles having the double representation are the orbicularis palpebrarum, the muscles of the vocal cords, the muscles concerned in

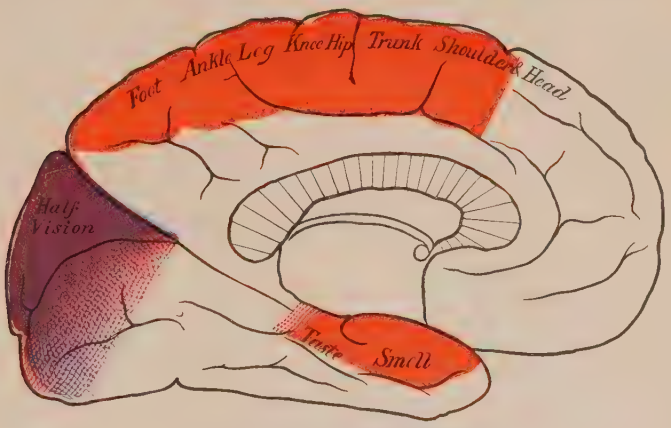


Fig. 166.—The Cortical Centres.

deglutition and in respiration. The muscles of the viscera and blood-vessels have no known representation in the cortex of the human brain.

The special sensations have a bilateral representation also; but the more specialized the sense, the less can one hemisphere take the place of the other.

Occipital, Parietal, and Temporal Lobes—Centres of Special Sense.—The special senses have two centres—the primary and the secondary. The primary centres are connected with the ganglia at the base of the brain; the secondary centres are situated in the cortex.

The *primary centre for vision* is in the posterior part of the optic thalamus, the external geniculate body, and anterior corpora quadrigemina. The secondary centre is situated in the occipital lobe, and particularly upon its mesial surface and in that part known as the cuneus. Each occipital lobe is the centre

for visual impulses from the corresponding half of the retina of each eye; for example, the left occipital lobe is the centre for vision of the left half of the retina of each eye. This relation is shown in the diagram (see Optic Nerve). Total destruction of both occipital lobes, or even of a considerable part of them if the destruction involves the median surface, will cause blindness. Destruction of one lobe causes only half-blindness or hemianopsia.

The *primary centre for hearing* is in the posterior tubercle of the corpora quadrigemina and the internal geniculate body. The secondary centre is in the cortex of the first and second convolutions of the temporal lobe. Destruction of one temporal lobe causes deafness in the opposite ear. This deafness, however, is not complete because the sense of hearing has a bilateral representation; each ear, in other words, sends fibres to the temporal lobes of each side, although more fibres cross over than go to the lobe of the corresponding side. The consequence is that the loss of one temporal lobe is in a measure supplied by the other (*vide* Aphasia).

The *primary centre for smell* is in the olfactory lobes. The secondary centre is probably in the uncus and in part of the hippocampal convolution. Whether the tracts for the sense of smell are connected with the optic thalamus or other ganglia is not definitely known.

The primary centre for taste is not known, but sensations of taste may connect with the optic thalamus before passing into the secondary centre, which is in the hippocampal convolution.

Centres for Memories.—There are certain classes of sensations and perceptions, simple in character and frequently repeated, so that they finally get to be used almost automatically in their work. These impressions relate to the use of the muscles in speech. in writing, and in gesture language, also to other frequently repeated purposeful movements of the limbs. The muscular movements in writing and speaking are so often repeated that certain areas in the cortex are set apart for the memories of these processes. The visual sensations and ideas elaborated from them, which are frequently repeated in learning to read, have also a centre which is set apart for them. This forms a centre for the visual memories of language. In the same way there are auditory sensations and ideas elaborated and so frequently repeated as to be used automatically in acquiring language. These are stored up as auditory memories. We have what may be called motor memories connected with speech and gesture. These special memories have been found to have a certain localization in the brain. The centre for the memories of the articular movements of speech is in the posterior part of the third left frontal convolution; the centre for the memories of the movements of

writing is not perfectly well known, but is thought to be at the posterior part of the second left frontal convolution. The centre for the memories of gesture language is unknown. The centre for the memories of ordinary co-ordinate movements is perhaps in the inferior parietal lobule. The centre for the visual memories of written language is in the angular gyrus, extending backward from there into the occipital lobe. The centre for the auditory memories of spoken language is in the posterior part of the first and the corresponding upper part of the second temporal convolution. In right-handed people all the memory centres are in the left cerebral hemisphere; in left-handed people they are in the right hemisphere. The destruction of these memory centres produces different forms of aphasia, as will be described later. In addition to that, disturbances in these centres are produced by lesions which cut off the associating fibres connecting these centres with each other or with motor or sensory centres proper.

The Centrum Ovale, Corpus Callosum, and the Associative Functions of the Brain.—The different parts and centres of the brain are connected together by the associating tracts and with lower levels by the projection fibres. The simpler and less developed centres of the two halves of the brain are closely connected by fibres that run chiefly in the corpus callosum. The more highly specialized and less simple in function a centre, the less close is its commissural connection and the more independent is one-half of the brain from the other. Thus the centres for the movements of the thorax in respiration are closely bound with each other; those for the purposeful movements of the hands less so; those for receiving visual impressions are almost independent; and the centres for the memories, which are still more highly specialized, are practically entirely independent. We infer that the higher mental functions, therefore, work either in one cerebral hemisphere or in the other, and that the two halves of the brain do not co-operate with each other in much of the higher intellectual work.

The corpus callosum is the great commissural tract connecting the two cerebral hemispheres and their respective centres. The anterior commissure does some of the same work, being more specially connected with the function of olfaction. The posterior commissure has comparatively few bilateral connecting fibres, its function being more to connect the thalamus with the cranial nerve nuclei and other centres below.

The Corpus Striatum.—This ganglion is in close relation with the cerebellum and with nuclei in the pons. It is also in connection with fibres that come up from the muscle-sense tract, in the spinal cord. Its functions are therefore probably connected with securing co-ordinate and purposeful movements. Destruc-

tion, however, of this ganglion in the human brain produces no definite symptoms, and local lesions of it cannot be diagnosticated. It is therefore called clinically a *latent region.*

The Thalamus Opticus.—The thalamus is in relation by its projection fibres with the frontal, parietal, occipital, and temporal cortex. The fibres that go to the occipital cortex are connected with the optic tract, and have to do with the function of vision. The fibres that go to the temporal lobe are connected with the auditory tract, and have to do with the function of hearing. The optic thalami seem to have some relation to the expression of emotions. In cerebral paralyses in which they are involved, the patient cannot involuntarily express joy, grief, etc. Lesions of the posterior part of the thalamus will produce partial blindness. Other than this, lesions of the optic thalamus produce no definite symptoms which enable us to make a local diagnosis. Disturbances of hearing have not certainly been traced to lesions in the thalamus. It is thought to be a primary centre for sensations of touch, muscular sense, and perhaps for smell and taste, but no definite facts in human pathology have as yet satisfactorily proved this. Lesions of the thalamus sometimes produce various forms of mobile spasm, but these are generally attributed to irritation of the fibres of the internal capsule, which go close to it. Hence, aside from disturbances of vision, the optic thalamus also must be considered clinically a latent region.

The Corpora Quadrigemina.—The anterior tubercles of the corpora quadrigemina, together with the external geniculate bodies, form part of the primary centres of vision. The anterior tubercles, however, have more to do with reflex movements of the pupil and ciliary muscles than with the actual visual function. The posterior tubercles of the corpora quadrigemina and the internal geniculate body are thought to be connected with the auditory nerve. They also appear to receive some fibres from the cerebellum; hence their injury or disease produces some disturbances in equilibrium and possibly in hearing. Owing to the fact that the nuclei of the third nerves and the red nuclei lie beneath the corpora quadrigemina, lesions of these latter produce irritations and paralyses of the third nerve, disturbances in equilibrium and forced movements. Lesions in this neighborhood sometimes cause somnolent and stuporous states.

The *red nuclei* are connected with the anterior cerebellar peduncles on the one hand and with the lenticular nucleus and optic thalamus on the other, and are concerned in securing equilibrium and the adjustment of the body in space.

The Cerebellum.—The cerebellum is connected with the pons, the cerebrum, and spinal cord. It sends impulses through the

middle peduncles to the gray matter in the pons, and thence down into the antero-lateral columns of the cord. It sends impulses to the olivary bodies, and thence up to the great ganglia at the base of the brain, and finally it sends impulses through the anterior peduncles, which cross over, enter the red nuclei, and connect with the thalamus, corpora striata, and perhaps with the cortex of the parietal lobe. It receives impulses from the cortex of the frontal, parietal, occipital, and temporal lobes, which go down into the pons, connect with nuclei there, and thence pass up into its hemispheres. It also receives impulses from the spinal cord, through the posterior peduncles. There is therefore a nervous circuit between the cerebrum, brain axis, and cerebellum: impulses pass down from the cerebral cortex to the pons, thence to the cerebellum; and other impulses pass up from the basal cord to the cerebellum. Having received these impulses, it discharges chiefly along the anterior peduncles into the basal ganglia, which act as its end-organ (Fig. 167). The cerebellar influences directly affect the spinal cord but little. The cerebellum has undoubtedly power of securing purposeful movements and of enabling us to keep our equilibrium and maintain our relations in space. The vermis or median lobe is the part which in man is most important in doing this work. Lesions of the lateral lobes or hemispheres produce few direct symptoms, and they are called latent regions. Injuries of the median lobe, however, produce disturbances in equilibrium, forced movements, and a peculiar form of inco-ordination in gait which is known as cerebellar ataxia. Lesions of the middle peduncles produce forced movements also, the forced movements being either toward or away from the side of the lesion, according as it is an irritating one or a destructive one.

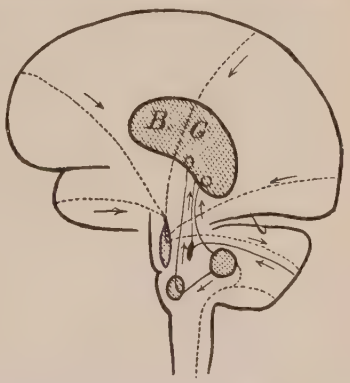


FIG. 167.—SHOWING THE RELATIONS OF THE CEREBELLUM TO ITS END-ORGANS, THE BASAL GANGLIA, AND TO OTHER PARTS.

The *pons varolii* contains some of the cranial nerve nuclei and collections of nerve cells which are connected with fibres from the cerebral cortex on the one hand and the cerebellum on the other. It also contains the long tracts of nerve fibres that pass from the cerebrum down through into the medulla and spinal cord and transverse tracts of fibres which connect the two hemispheres of the cerebellum. Lesions in it cause disturbances in

function of the cranial nerves and of the motor, sensory, and commissural tracts.

The *medulla oblongata* contains centres of the cranial nerve puclei, and in it also are various reflex and automatic centres controlling and regulating the vasomotor system, respiratory and cardiac rhythm, visceral movements and secretion.

The *olivary bodies* are connected with the cerebellum and basal ganglia, but not with the spinal cord. Impulses pass to these bodies from the cerebellum, thence up to the brain. When injured, disturbances of equilibrium and co-ordination occur.

The Latent Regions of the Brain.—There are certain parts of the cerebral cortex destruction of which and irritation of which produce no special and distinctive phenomena in man. These are the greater part of the temporal lobe of the right side and a portion of the temporal lobe on the left side. A part of the inferior parietal lobule also may be regarded as a latent region. The frontal lobe we have already spoken of as being concerned with certain mental functions, but lesions here often produce no symptoms, and they may be to a certain extent regarded as latent. The corpora striata, optic thalami, portions of the centrum ovale, and the two lateral hemispheres of the cerebellum are latent areas.

Brain Weight.—The average weight of the male brain is 1,358 gm.; that of the female, 1,235 gm. The weight varies with age, sex, race, and intelligence, and with a number of other factors. The average weight of the brain at birth is 327.8 gm.; the brain grows rapidly until the age of four, then more slowly until the age of seven, then very slowly up to the age of sixteen to twenty. At about the age of forty-five in man and fifty in woman it begins to lose weight slowly, and at the age of eighty or over it has lost about 120 gm. (4 oz.). The brain of man weighs absolutely about nine per cent more than that of woman. Relatively to the body weight, the brain weight of man is about 2%; that of woman a very little less. The sexual difference is extremely small.*

The brain weighs more in the civilized races, and more in certain of the civilized races than others; the brains of English, German, and Scotch weigh more than those of French, Italian, and Russian. Some of the African and Australian tribes have the smallest brains, the average negro brain weighing 1,250 gm. When a brain weighs less than 1,130 gm. in man or 990 gm. in woman, it is called a microcephalic brain; if the weight is above 1,490 gm. in man or 1,345 gm. in woman, it is called a megalocephalic brain.

Brain weight has a certain relation to intelligence, which is not, however, an absolute one. Among a hundred men of more than average intelligence, the percentage of large brains would be about 25, whereas the percentage of large brains among persons of ordinary or low intelligence would be not more than 4 or 5. In estimating the importance of brain weight, one must consider the height, the weight or volume of body, muscular mass, and

* J. C. Brown finds that after making all allowances, woman's brain weighs about one ounce less than man's.

superficial area; these are called the somatic factors. The following formula has been devised by Snell for estimating the mental power of different animals:

$$P = \frac{H}{K^s}$$

In this formula P represents the psychical factor or the amount of intelligence, H the brain weight, K the body weight, S the somatic factor. The somatic factor has been estimated to be for mammals about 0.666. Applying this formula, we find that, expressed relatively, the intelligence of man equals 0.87; woman, 0.86; the ape, 0.42; the rabbit, 0.59; the birds, from 0.167 to 0.09.

The relative weight of different parts of the brain is about as follows: Frontal lobes, 28%; parietal lobes, 36%; occipital, 10%; temporal, 13%; lobus caudicis or island of Reil, 9%; pons, 1½%. The cerebellum weighs about one-eighth as much as the cerebrum. The proportion of the gray to the white matter in adults is 60 to 40 (Vierordt).

The depth of the primary fissures is not quite an inch (20 to 23 mm.).

There are one thousand two hundred to two thousand million cells in the cerebrum, and about ten million large cells in the cerebellum (Meynert).

About one million cells to a square centimetre is the estimate of Engel.

Preserving and Cutting the Brain.

The brain should be placed in a gallon of a 2½-per-cent solution of bichromate of potash. This must be changed daily for a week, then twice weekly for a fortnight; then it should remain in the solution for three or four months, a few crystals of thymol being added. After about three months place the brain in 95% alcohol. In a few days it will be ready for cutting.

In cutting the fresh or preserved brain for the purpose of locating gross lesions, remove the pons, medulla, and cerebellum, place the brain on its base, and make sections in accordance with the directions (Fig. 168). The sectional views exposed are shown in the following series of cuts, which are based upon those of Exner.

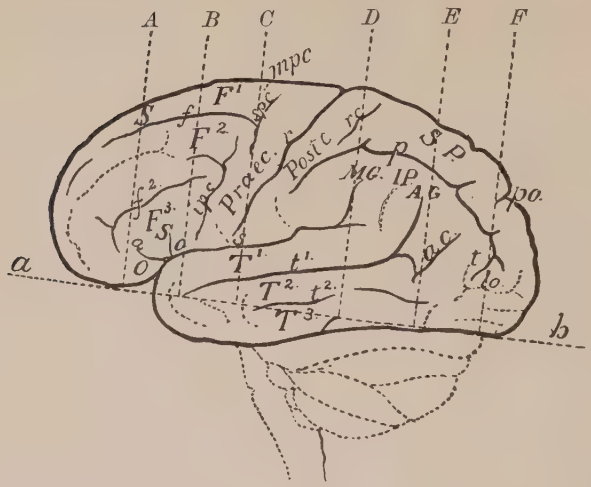


FIG. 168.—SHOWING THE POINTS AT WHICH THE SECTIONS ARE MADE. *a*, *b*, Horizontal line from base of frontal to base of occipital lobe; A, vertical section through middle of third frontal convolution; B, through operculum, *o*; C, through superior præcentral and lower end of Rolandic fissures; D, through *rc* and posterior end of Sylvian fissure; E, through angular gyrus and anterior occipital fissure; F, through parieto-occipital fissure. B should be about half-way between A and C; E half-way between D and F. (See Figs. 169-174.)

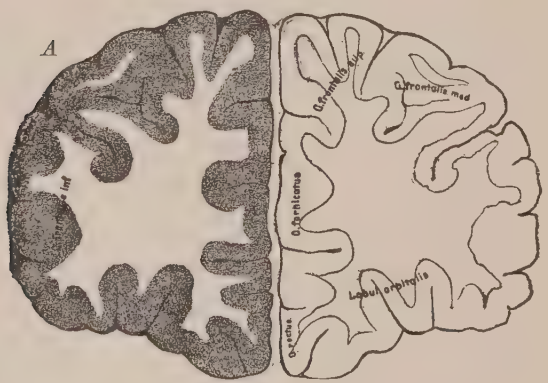


FIG. 169.—SECTION THROUGH LINE A, FIG. 168.

FIG. 170.—SECTION THROUGH LINE B, FIG. 168.

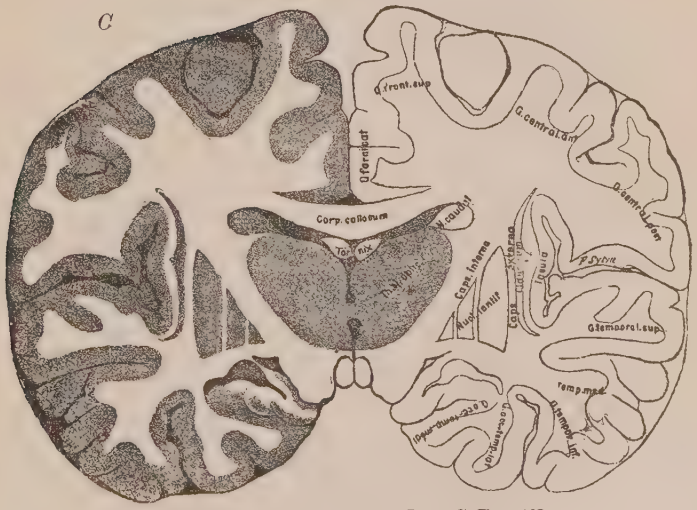


FIG. 171.—SECTION THROUGH LINE C, FIG. 168.

D

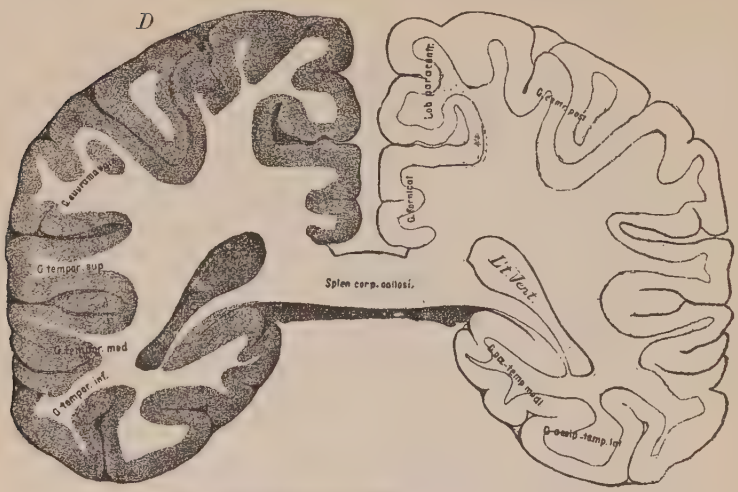


FIG. 172.—SECTION THROUGH LINE D, FIG. 168.

E

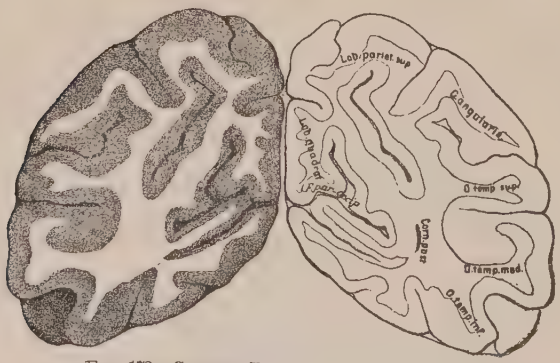


FIG. 173.—SECTION THROUGH LINE E, FIG. 168.

F

FIG. 174.—SECTION THROUGH LINE F, FIG. 168.

CHAPTER XVII.

DISEASES OF THE BRAIN AND ITS MEMBRANES.

General Symptoms.

It will add to the intelligibility of descriptions of brain diseases and their symptoms if one first makes himself familiar with certain general symptoms that underlie more or less nearly all organic disorders of this organ. Symptoms due to disease of the brain may be placed in four classes: first, general symptoms of brain irritation; second, general symptoms of brain pressure; third, symptoms of focal irritation or destruction; and, last, those due directly to the pathological process itself.

The symptoms of brain irritation are headache, vertigo, vomiting, photophobia, mental irritability, insomnia, peculiar feelings of fulness and pressure about the head, noises in the ears or in the head, tenderness about the scalp, and in severe cases convulsive symptoms and delirium.

The symptoms of brain compression are headache, vomiting, mental hebetude or dulness, perhaps some form of paralysis, contracted pupils, and eventually coma. With this there is often constipation and retracted abdomen.

The symptoms of brain irritation are often, perhaps usually, associated with a hyperæmia. The symptoms of brain compression may be associated with anæmia or œdema, and often in states of malnutrition, where the brain is impoverished, the symptoms resemble much those of compression. Pressure symptoms and irritative symptoms often lap one into the other, and they cannot always be sharply distinguished.

Focal symptoms depend almost entirely upon the location of the particular lesion. If it is in a motor area, focal symptoms of irritation would be spasmodic phenomena, such as convulsions. If the lesion were destructive, the symptoms would be those of paralysis or anæsthesia.

The symptoms due directly to the pathological process itself may be very slight. Thus in case of a tumor of the brain, the symptoms are mainly caused by pressure, irritation, and local disturbance of certain special parts of the brain. In suppuration, however, the process itself may produce general symptoms such

as are associated usually with sepsis—chills, irregular fever, mental hebetude, prostration, emaciation, and sweats.

Among the symptoms produced by focal lesions there are a few which deserve some preliminary general study because they may be caused by lesions of very different kinds and occur consequently in very different forms of diseases. Those symptoms which we wish particularly to study here are hemiplegia and aphasia. These represent the two great dominating symptoms pertaining on the one hand to motor disturbance and on the other hand to sensori-motor disturbance.

Hemiplegia.—Hemiplegia is a paralysis of one-half of the body, involving the side opposite the lesion. The face, arm, and leg are usually all paralyzed; the arm most, the leg next, the face least. Hemiplegia may be either acute in onset or slow and progressive. Acute hemiplegia is the result usually of hemorrhages and softenings of the brain, more rarely of inflammations and injuries. Progressive hemiplegia begins gradually, as its name implies, and slowly increases until the height of the disease is reached. It is usually caused by tumors growing in one side of the brain, but it may be caused by a slowly developing patch of sclerosis, which sclerosis may be in turn only a part of a multiple sclerosis. Further description of the peculiarities of hemiplegia will be given under the head of Special Diseases of the Brain.

Aphasia.—Aphasia is a disease of the faculty of language; and it has a number of varieties, in accordance with the particular part of the brain involved and the particular portion of the mechanism of this faculty that is destroyed. By the faculty of language we include the processes by which we hear, see, and at the same time appreciate the meaning of symbols. It includes also the faculty of expressing to others, by voice, writing, or gesture, our ideas. It has therefore a receptive side and an emissive side. We may have lesions in the brain which destroy that part of the language faculty concerned in our power of seeing and understanding written words or the gesture language. In reading understandingly one sees certain words; these words revive certain visual memories connected with past perceptions. Thus one sees the word "book;" this suggests to him past memories of form, color, tactile and other sensations associated with the past perceptions of books. There is a certain centre in the brain where these visual memories for letters and words are located. When this centre is destroyed the memories are destroyed, and the word "book" or any other written word conveys no meaning. The patient can spell out the letters, he can see the letters, but he cannot read any more than if he had never been taught. The condition is known as *alexia* or *word-blindness*. Again, a person may have learned to associate certain gestures with definite ideas,

as the motion of carrying a glass to the mouth with that of drinking, or the motions of using a knife and fork with that of eating, or the motions of the deaf-and-dumb alphabet with certain words and ideas. These memories of gesture language are located in certain regions; and when they are destroyed the patient is no longer able to understand gestures or the sign language. This condition is known as *apraxia*. Both apraxia and alexia are forms of aphasia which come under the general head of *mind-blindness*. A person hears certain words, as, for example, the word "knife." This conveys to him a certain idea of the form, color, and other properties associated with knife. The memories associated with the auditory perception of different words are stored up in a certain locality which is the centre for auditory memories. When this centre is destroyed the person hears spoken words, but they convey to him no meaning. All that it said to him sounds as if it were in a foreign language: he hears, but he does not understand. This condition is known as *word-deafness*. So much for the receptive or sensory side of language.

In communicating our ideas, we speak, write, and make gestures. In speaking we make use of the organs of articulation, and this use involves the fine adjustment of a delicate muscular apparatus. In the act of expressing ideas we have to bring into play the memories of the past muscular movements of this articulatory mechanism. These movements were learned by a slow and painful process during infancy. After the power of speech is acquired, the mechanism works readily and almost automatically because we only have to send a stimulus to the centre which presides over the stored-up memories of the impulses to properly innervate the mechanism of speech. There is, therefore, a centre for the memories of the movements of articulation —a centre which is of course closely connected with the motor areas that directly innervate the larynx, pharynx, and oral and facial muscles. When a lesion destroys this centre for speech memories, a person is unable to reproduce the words necessary for expressing an idea; for example, he sees a knife, he knows what it is, but the memory of the motions necessary to express the word "knife" is gone. To him it seems that the name is gone, and that is the common way of expressing it. He cannot say the word "knife." The patient may wish to express the idea of pain. He feels the pain, he knows that he has pain, but he cannot revive those motor memories which are concerned in expressing the word "pain;" he cannot tell, therefore, in words what is the matter with him. When a person is thus troubled, he is said to have a form of motor aphasia for which the particular name given is *aphemia*. In the same way there is a centre for the memories of the muscular movements concerned in writing; and when a lesion

destroys this centre the patient is unable to write, though he may be able to speak. This condition is called *agraphia*. Finally there is a centre, less well defined, for the memories of the movements used in gesture language, and when this is destroyed the person is unable to express his ideas by gesture or sign language. This condition is known as *amimia*.

To resume now, we have the following divisions of aphasia:

Sensory Aphasia.		Motor Aphasia.
Mind-blindness.	Alexia.	Aphemia.
	Apraxia.	Agraphia.
Mind-deafness.	Word-deafness.	Amimia.
	Music-deafness.	
	Paraphasia.	

There is also a form of aphasia in which the patient misplaces words, often to an extent that makes his speech somewhat incoherent. This is called *paraphasia*, and is due to lesions which destroy some of the associative fibre-tracts connecting the special centres for language. Practically we often find mixtures of motor and sensory aphasia, or we have a motor aphasia with paraphasia. Patients may also lose the power of expressing themselves by ordinary speech, but may be able to use signs; they may not be able to write voluntarily, but can write to dictation; they may not be able to read directly, but can do so when they trace out the letters with a pen. These different forms are due to lesions which destroy various connecting tracts as well as certain of the centres. The commonest form of aphasia is the motor aphasia in which there is aphemia and agraphia; that is, loss of power to speak and write. This may not be complete, and then there will be with the motor aphasia some paraphasia.

In examining a patient who has aphasia one should note the following points: Whether he can speak, name objects, repeat words after you, write from dictation, copy; whether he can sing or express himself by gestures; whether he can read, spell, read by tracing letters with the pen, read words that he has written himself; whether he can understand spoken language or gesture language; whether he can appreciate the nature and use of objects. At the same time he should also be tested for hemiplegia, deafness in each ear, acuity of vision, hemianopsia, and hemianæsthesia.

The location of lesions in the various forms of aphasia has been made out with considerable definiteness. The lesion in aphemia is in the posterior part of the third left frontal convolution or Broca's convolution; for agraphia, sometimes in the base of the second frontal convolution, sometimes in the angular gyrus. For gesture language there is no known centre. The

lesion in word-blindness and apraxia is in the angular gyrus, extending back into the occipital convolution. The lesion in word-deafness is in the posterior part of the first temporal and upper part of the second temporal convolution. In right-handed people the lesions are always on the left side; in left-handed people on the right side (see Fig. 165).

Malformations of the Brain and its Envelopes.

Congenital malformations of the brain are of little practical importance, for in most cases the monsters cannot live and in all

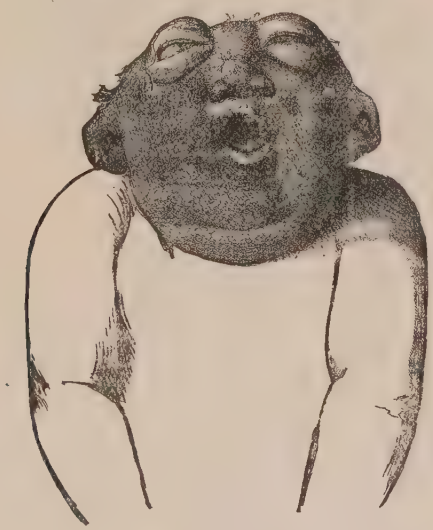


Fig. 175.—Acrania.

cases they are better dead. I shall simply give a brief enumeration of the important forms.

Abnormalities of the brain.	Anencephaly.
	Porencephaly.
	Absences or malformations of parts, *e.g.*, Cyclopia.
Abnormalities of brain and its envelopes.	Acrania.
	Meningocele.
	Encephalocele.
	Hydrencephalocele.

Anencephaly is always present with acrania. In anencephaly the cerebellum and part of the basal ganglia may be present. In such case the child can live a short time (Fig. 175).

Porencephaly is often an artificial condition. It will be described later.

In cyclopia there is an undivided anterior cerebral vesicle;

the orbits form a continuous cavity with a single rudimentary eye (Fig. 176).

In acrania there is usually anencephaly.

Meningocele is a hernia of the brain membranes, arachnoid and dura through a cleft in the skull. In encephalocele the

Fig. 176.—Cyclopia.

brain also protrudes. Both these forms occur usually in the occipital region and almost invariably in the median line. In hydrencephalocele there is a sac with fluid contents.

Diseases of the Membranes of the Brain.

The diseases to be considered under this head are anæmia and hyperæmia, inflammation of the dura mater or pachymeningitis, and inflammation of the pia mater or leptomeningitis.

Anæmia and Hyperæmia of the Membranes of the Brain.—Anæmia of the membranes of the brain is a condition that cannot be separated from anæmia of the brain substance, and will be considered in connection with it. Hyperæmia of the brain membranes, so far as it relates to hyperæmia of the pia mater, must also be considered in connection with hyperæmia of the brain tissue. Dural hyperæmia, or congestion of the dura mater, is a condition which occurs as the result of injuries, sunstroke, and of certain infective poisons, especially that of syphilis. The symptoms are those of pachymeningitis of the slight

grade, and will be described under that head. They consist mainly of pain, occasional attacks of vertigo, and sensations of fulness about the head. The treatment is that for the beginning stages of a meningitis.

Inflammation of the Dura Mater or Pachymeningitis Externa.—It has been the custom to describe two forms of pachymeningitis, the external and the internal. Internal pachymeningitis, or hæmatoma of the dura mater, is properly a hemorrhagic disorder and is described under the head of Dural Hemorrhages. A true inflammation confined to the internal surface of the dura alone is of extremely rare occurrence.

Pachymeningitis externa is a disease that involves, at first at least, the outer surface of the dura, and is usually of surgical origin and interest.

Etiology.—Accidents, injuries, caries of the petrous bone in mastoid disease, of the ethmoid bone in ozæna, necrosis, syphilis, and erysipelas are the usual causes.

The *symptoms* are local headache, fever, delirium, sometimes even convulsions and paralysis. In the severe cases the disease has usually extended and involved the pia. Pus is generally formed, and burrows between the bone and dura. The disease is recognized mainly by the discovery of the local cause.

The *course* is acute or subacute.

The *treatment* is a surgical one.

Inflammation of the Pia Mater or Leptomeningitis.—Inflammation of the pia mater has the following types: simple meningitis due to some infection, epidemic cerebro-spinal meningitis due to a specific general infection, tubercular meningitis, and syphilitic meningitis.

All these forms of meningitis may be either acute or chronic; the chronic form being usually simply a sequela of the acute.

Acute Simple Leptomeningitis.—*Etiology.*—Acute leptomeningitis is always due to an infective process reaching the cerebral membranes usually directly from without, but sometimes through the blood. The so-called œdematous inflammation of alcoholism and inanition is the only exception. The most common source of infection is disease of the middle ear and mastoid cells. Disease of the frontal sinuses and upper nasal passages; operations on those parts; disease, injuries, and fractures of the cranial bones—are also common causes. Pneumonia is the most frequent infective disease in which the pyogenic organisms are carried by the blood. After this come pyæmia, septicæmia, variola, scarlet fever, more rarely endocarditis, empyema, rheumatism, measles, typhoid fever, and mumps. Occasionally a brain abscess reaches the surface and sets up a meningitis. Insolation can of itself not cause it. The disease is more frequent in males,

and is distributed through all ages of life, though it occurs oftener in the young.

Symptoms.—The symptoms in the various types differ somewhat, but have a general similarity. They are to be broadly grouped into the prodromal, the irritative, the depressive, and the paralytic stages.

Prodromal symptoms are shorter and less marked in simple meningitis than in tubercular. The patient suffers from malaise, languor, headache, vertigo, irritability, loss of appetite, and vomiting. Of these symptoms headache is the most notable.

In the second stage the dominant symptoms are headache, delirium, rigidity of the neck, hyperæsthesia of the skin, retraction of the abdomen, vomiting, irregular fever, contracted and often unequal pupils, sometimes optic neuritis or retinitis. The headache is usually persistent, with exacerbations of great intensity. Rather early in this disease the patient's mind begins to wander; he mutters incoherently; he may have periods of violence alternating with stupor. In some cases there is a continuous low-muttering delirium. Vomiting also occurs early and is of a violent, explosive (projectile) character. This symptom is not always present. The head is bent back and the patient can be lifted from the pillow by placing the hand under the occiput. There is sometimes a general rigidity which resembles catalepsy. Drawing a dull point along the skin causes a red line to appear (*tache cérébrale*). Pinching or rubbing the skin causes much pain. The abdomen falls in and assumes a characteristic "boat-shape." The pupils are usually contracted and uneven. The eyes are intolerant to light. Optic neuritis occurs often when the inflammation is at the base; but it is a late symptom. Convulsions and local paralyses of the cranial nerves, causing sight strabismus, ptosis, or facial palsy, may occur. The fever is irregular in course and not high—101° to 103°. The pulse is usually irregular or rather intermittent. It varies greatly in frequency and may be rather slow—50 to 70. Respiration is rather quickened and sometimes irregular. The bowels are constipated; the urine small in amount and sometimes albuminous.

In the paralytic stage the patient becomes stupid or comatose; there is still some rigidity, except in the very last stages. The abdomen is still greatly retracted, the pupils may now dilate, the skin become moist, and the patient's bowels and bladder move involuntarily. Death now occurs in one or two days as a rule.

When the disease is mainly on the convexity of the hemispheres there is more delirium, convulsive and paralytic troubles; when confined to the base there is less delirium, while paralysis of cranial nerves, optic neuritis, vomiting, and retraction of the head are commoner or more prominent.

Course and Duration.—The disease may begin suddenly, and the patient pass at once into the comatose state, dying in a few days. Usually the process lasts one or two weeks; it may be prolonged for several weeks.

The *prognosis* is very grave, but it is less serious than in tubercular meningitis and more serious than in the cerebro-spinal form.

The *diagnosis* is based on the presence of an exciting cause, such as disease of the ear or nose, trauma, infective fevers, and upon the presence of the symptoms given. It is usually easily recognized, the main difficulty being to distinguish it from tubercular and cerebro-spinal meningitis.

Pathology.—The disease is a fibro-purulent or purulent inflammation. It involves usually the base more than the convexity, but the reverse may happen. The ventricles are often involved and may be independently inflamed. There are descriptions, therefore, of simple basilar meningitis, meningitis of the convexity, and ventricular meningitis or ependymitis. The inflammatory deposits are most conspicuous along the course of the Sylvian fissure and the vessels branching from it, about the optic chiasm, and at the posterior and under surface of the cerebellum and the sides of the pons. It may lie only in the subarachnoid cavity, but usually the arachnoid and sometimes the dura are implicated. There is increase of fluid in the ventricles and arachnoid cavities, and this fluid may be turbid. The surface of the ventricles may show an inflammatory process.

The micro-organisms found in meningitis are the pneumococcus, streptococcus pyogenes, intracellular diplococcus, the pneumo-bacillus, and a bacillus resembling that of typhoid fever. Still others have been described, and the process is apparently a mixed infection, though the pneumococcus is found oftenest.

Treatment.—Prophylaxis is the most important measure, as there is no specific treatment. Chronic disease of the ear and nasal sinuses should be attended to, and injuries of the skull treated with the strictest regard to antisepsis. The patient should be kept quiet, a dose of calomel given, and small doses of iodide of potassium administered at frequent intervals. An ice cap may be applied to the head and hot applications to the feet. Hot poultices along the upper spine are useful. Opium must be given for the pain, if needed; and antipyretics or phenacetin sometimes answer, in a measure. The internal use of iodoform has been highly recommended, gr. vi. to gr. xij. daily; shaving the head and rubbing upon it an ointment containing 20% iodoform, then covering the scalp with an oiled-silk cap, is a treatment highly spoken of. Surgical intervention is sometimes justifiable.

Epidemic Cerebro-Spinal Meningitis (Spotted Fever).—This is an acute infective disorder and is produced by a special micro-organism. It has certain peculiar clinical characteristics which lead us to describe it separately. Anatomically the changes involve the spinal membranes as well as the cerebral.

Etiology.—The disease most frequently attacks children, but it may occur at any age. Males are affected rather more often than females. It prevails in the form of epidemics which affect cold and temperate climates especially, and which travel from one part of the country to another. It may occur sporadically. It most frequently develops during the winter season, and attacks persons who are living in crowded houses, tenements, or barracks. It is slightly contagious. One attack does not confer an immunity against a second.

Symptoms.—The general appearance of a person attacked with the disease is that of one who has been poisoned by some agent which is extremely prostrating to the whole system and at the same time one which has a specific inflammatory effect upon the meninges of the brain and spinal cord. When the disease is rapid and malignant, the patient seems to die of an acute toxæmia before any inflammatory process has time to develop. In milder cases and those of longer duration the prostration is less, and the evidences of inflammation of the meninges then develop in the typical way. The disease may begin with prodromal symptoms of malaise, discomfort, pain in the neck, vomiting, and headache. As it develops, the headache, accompanied with giddiness, increases, pain and stiffness in the neck become more violent, pains run down the back and radiate to the limbs; there is photophobia, and delirium in many cases is marked. The skin is hyperæsthetic, the pulse rises to 120 or higher, the temperature varies very much and is usually raised to 103°, 104°, or even more. The bowels are generally constipated. In most cases there develop certain skin eruptions, usually in the form of purpuric spots; herpes, urticaria, and erythema are occasionally seen. These eruptions vary very much in different epidemics; the purpuric spots are the most important from a diagnostic point of view, and have given to the disease the name of *spotted fever*. As the disease progresses the symptoms of irritation and pain give way to those of somnolence, stupor, and paralysis. Optic neuritis, acoustic neuritis, and inflammation of other cranial nerves take place, and paralyses of the limbs may be added.

The disease may run a short and malignant course, killing the person in a few hours or one or two days. In moderate cases it lasts about two weeks. A large number of different varieties of the disease are described, such as the abortive form, fulminating form, and typhoid form. The disorder is often complicated with

pneumonia and bronchitis, less often with inflammation of the joints and serous membranes. The disease often leaves very serious sequelæ, the most important being deafness and spinal irritation or chronic spinal meningitis. A large proportion of deaf-mutes owe their affliction to this disease.

Pathological Anatomy.—In the very acute cases the post mortem shows nothing but the evidence of very severe blood-poisoning. In the milder and more chronic cases an inflammation involving the pia and arachnoid of the brain and cord is found. This inflammation is fibrinous or fibro-purulent in character, and may be accompanied with the exudation of a good deal of inflammatory material. Bacteriological researches show that this disease is due to the presence of a specific micro-organism which is apparently very much like that which causes pneumonia.

The *diagnosis* is based upon the history of an epidemic of the disease being present, upon the presence of the ordinary symptoms of acute cerebral and spinal meningitis, such as headache, delirium, retraction of the head, the sunken abdomen, hyperæsthesia and pains; finally, the presence of the peculiar purpuric spots or of herpes of the face will enable one to make a positive diagnosis. One must learn to distinguish the disease from typhus, tetanus, uræmia, pneumonia, and from the other forms of meningitis, especially the tubercular. The diagnosis is often made difficult by the fact that cerebro-spinal meningitis may occur in a sporadic form, and it is well known that after a community has been once visited with an epidemic, these sporadic cases are liable to crop up from time to time for many subsequent years. The sudden onset of the disease, the spinal symptoms, the skin eruption, the absence of history of injury or of evidence of tuberculosis will usually enable one to recognize the disorder.

The *prognosis* varies much with the epidemic, but the disease is always a serious one. The mortality ranges from twenty to eighty per cent; it is worse when the disease comes on suddenly and severely, with early coma. It is better in persons over the age of ten. Cranial-nerve complications are unfavorable, in that they are apt to leave permanent deafness. Severe spinal complications are apt to leave their mark in the form of a chronic meningeal trouble.

Treatment.—There is no specific remedy for the disease, and the ordinary antiphlogistic measures such as mercury and iodides are of less value than in other forms of meningitis. The patient should be given sustaining food, and everything possible should be done to counteract the depressing effects of the toxæmia. Opium or morphine internally, chloral, digitalis, quinine, benzoate of sodium and salicylate of sodium, and alcohol are the

drugs which have been specially recommended. Warm baths, hot moist applications, and leeches have all been tried with more or less good results.

Tubercular Meningitis (Acute Hydrocephalus).—This is a form of meningitis due to infection with the bacillus tuberculosis. It differs pathologically from other forms in the character of the infective organism; anatomically, in the fact that the inflammation is usually and chiefly basilar and never purely purulent; etiologically, in that it chiefly affects young children; and symptomatologically, in the presence of prodromata and a more irregular course.

Etiology.—Tubercular meningitis occurs chiefly between the ages of two and ten, sometimes in infancy, rarely in adult life, very rarely after the age of fifty. Males are rather more subject to it. A hereditary history of phthisis, a scrofulous diathesis, bad hygienic surroundings, and the presence of tuberculosis elsewhere in the body predispose to it. Tuberculous milk, the eruptive fevers, especially measles, blows on the head, and great emotional excitement appear to act as exciting causes.

Symptoms.—A knowledge of the prodromal symptoms is especially important. These are paroxysmal and intensely severe headaches and darting pains in the head, vertigo, loss of appetite, explosive vomiting without nausea, the vomited matter being usually colorless and watery, constipation, an altered disposition, and irritability. The *tache cérébrale* or cerebral macule, more rarely ptosis and facial paralysis, may appear early. The prodromal stage often lasts, with remissions, three or four weeks. When the disease sets in there is more persistent headache; vomiting, fever, and the other symptoms of meningitis already described appear. The irritative stage gradually passes into the paralytic and comatose. Death occurs in two or three weeks. In infants the disease often runs a very obscure course, the patient showing chiefly symptoms of brain compression.

Pathological Anatomy.—In rapidly fatal cases, with severe symptoms, there may be only an intense congestion of the brain with numerous miliary tubercles in the pia mater at the base and over the convexity. Here we must assume that a bacillary toxine causes the symptoms. In most cases there are decided deposits of tubercles at the base, with fibrinous inflammatory deposits about the optic chiasm, along the fissure of Sylvius, at the sides of the pons and elsewhere. Miliary tubercles are seen scattered over the convexity and in the choroid plexus and ventricles. They are generally found in the spinal membranes also, especially over the cauda equina. The tubercles lie beneath the pia surrounding the small vessels. They may coalesce into large tubercular nodules. There is usually an increase in the arachnoid

fluid, and in most cases an increase in the ventricular fluid. Somewhat rarely there is very great distention of the ventricles and compression of the convolutions. This condition used to be called *acute hydrocephalus*. Small spots of softening may be seen from obliteration of the vessels by the tubercles. The bacillus tuberculosis is found in the tubercular nodules.

Diagnosis.—As regards the form of the disease, this is based on the hereditary history, the age, the existence of tuberculosis of the lungs or other organs, and the peculiar prodromata of the disease. Occasionally tubercles can be seen on the choroid.

Prognosis.—This is usually absolutely bad, yet post-mortem observation of cases dying with practically no inflammatory change makes it seem possible that the disease might be checked, and a good many cases are reported where it apparently has been. Some of these are, however, probably cases of hereditary syphilis.

Treatment.—So far as now known, this is not different from that given under the head of meningitis elsewhere. It seems, however, as if in time some anti-toxine may be discovered which will check the progress of the poison and the development of the tubercle; meanwhile the best thing to do is to give small doses of iodide of potassium at frequent intervals and use symptomatic treatment.

Chronic Hydrocephalus.

This is a disease mainly of infancy, characterized by a gradual enlargement of the head, with mental deficiency and symptoms of brain irritation caused by an accumulation of fluid in the ventricles of the brain.

The old term "acute hydrocephalus" meant an acute inflammation with effusion, but the name is not needed and is best dropped. Chronic hydrocephalus is not an inflammatory process, but one due to mechanical causes or to defects in structure or nutrition. The fluid always accumulates in the ventricles of the brain; hence chronic hydrocephalus is always *internal*. The so-called external forms of hydrocephalus are inflammatory or else are secondary to meningeal hemorrhage or brain atrophy. Chronic hydrocephalus is almost always a disease of infancy and is generally congenital. It may, however, be acquired. In speaking of chronic hydrocephalus, we refer to the chronic internal congenital disease.

Etiology.—Four out of five cases begin at birth or within the first six months of life. Syphilis (J. Lewis Smith), alcoholism, lead-poisoning in the parents, and some unknown family taint predispose to the disease. Poverty and poor nutrition and rickets are also factors.

Symptoms.—The head may be so large at birth that instrumental help is needed. More often the parents notice a gradual increase in the size of the child's head, beginning soon after birth. The forehead bulges, the occiput stands out, the fontanelles and sutures widen, and pressure shows evidence of fluctuation. Meanwhile the face does not grow much and the result is to give a triangular shape to the head. It may measure twenty-four, twenty-seven and one-half (Minot), thirty-two (Bright), and even forty-three inches (Klein) in diameter. These extreme measurements are reached only after one or two years. With this abnormal growth of the head, mental and physical symptoms appear. The infant is restless and irritable, its appetite may be good, but the general nutrition is poor and its bodily growth is retarded.

Fig. 177.—Chronic Hydrocephalus.

The mind does not develop; usually it does not or cannot learn to walk. It may be unable to support the weight of its head. There is strabismus and sometimes optic atrophy. The pressure of the dropsy thins the orbital bones and forces down the axis of the eyeballs (see Fig. 177). Vomiting, coma, and convulsions eventually appear, and the child dies of exhaustion or some intercurrent disease in two or three years.

In some cases the trouble is less serious, it ceases to progress, the bones solidify, and the child grows up with good intelligence.

Chronic hydrocephalus sometimes develops in late childhood and in adult life. It is then due to some tumor or inflammatory process obstructing the venæ Galenæ and the aqueduct of Sylvius. The symptoms are chiefly those of brain pressure, and the disease cannot be recognized with certainty.

The hydrocephalus which is associated with the brain atrophy

of insanity and old age or with general dropsical conditions has no kinship with the process we are now describing.

Pathology.—The disease is due to the gradual accumulation of a serous fluid in the ventricles of the brain. The cause of this is an inflammatory or developmental obliteration of the foramen of Magendie and the adjacent lateral foramena of Mierziejewski. This prevents the escape of the ventricular fluid into the general arachnoid cavity. Contributing factors are congenital or acquired defect in the absorbents of the ventricles and a rachitic and easily yielding skull. The attempts to make chronic hydrocephalus an inflammation are failures, though there is at times a thickening of the lining membrane of the ventricles. The lateral ventricles are principally and often solely affected, and these are so distended as to press out their cerebral walls, flattening the convolutions, and turning them into a thin shell often less than a quarter of an inch in thickness. Sometimes only one lateral ventricle, and in rare cases only the fourth ventricle, is affected with the dropsy.

The *diagnosis* has to be made from rickets and an acute inflammatory process. In rickets the head is square, the fontanelle does not bulge, the enlargement is less, and there are signs of the disease in the bones elsewhere.

Prognosis.—The congenital cases usually die in a few months, or at least before the third year. Those developing in infancy may live for four to six years; and in mild cases the disease ceases to progress and a fairly healthy adult life is reached.

Treatment.—A great many measures have been recommended, but there is no unanimity about any one of them. In such a state of therapeutics it is safe to say that treatment is of little use. The most rational measure is the inunction of mercury and the administration of iodide of potassium combined with tonics. Surgical measures such as tapping the ventricles are irrational and need not be discussed. Quincke's method of tapping the spinal canal will not apply in these cases, though it is a practicable measure as I have found by experiments on the cadaver. Strapping the head with diachylon plaster is recommended by Trousseau and by J. Lewis Smith.

CHAPTER XVIII.

DISEASES OF THE BRAIN.

These diseases, like those of other parts of the nervous system, consist of malformations, vascular disturbances, inflammations, softenings, hemorrhages, degenerations and scleroses, chronic infections, tumors, and functional disorders.

Cerebral hyperæmia is a condition in which there is an excessive amount of blood in the cranial cavity; it may be acute or chronic, active or passive.

Etiology.—In the description which is to follow I shall refer only to those conditions of hyperæmia of the brain which are pathological. It is a well-recognized fact that hyperæmia of the brain occurs physiologically under excitement and over-activity of the heart and from various stimuli; but a pathological condition of acute congestion may be induced by sunstroke, certain drugs such as alcohol, and by injuries; also by mechanical causes which prevent the exit of the blood from the cranium. An acute congestion also occurs in mania and in many forms of fevers, as well as in the initial stage of meningitis. A chronic cerebral hyperæmia may be induced by the causes already mentioned as bringing on acute congestion. The prolonged use of alcohol, prolonged mental excitement, overwork, and worry may also lead to this condition. The foregoing causes lead to what is known as active congestion, in which the blood is driven in excess into the brain through the arteries. A passive congestion may exist in which the blood is prevented from leaving the brain and is kept mainly in the intra-cranial veins. The causes of passive congestion are mainly mechanical, such as cardiac disease and mechanical obstructions about the neck from tight clothes and an obstruction to the flow of blood from the lungs by playing on wind instruments.

Symptoms.—A great deal has been written regarding the symptomatology of cerebral hyperæmia, but many of the statements made are nothing but guesswork. Probably the main symptoms produced by an active congestion of the brain are a sense of fulness and pressure, a feeling of constriction about the head, some headache which may be vertical, mental excitement or irritability, confusion of ideas, vertigo, insomnia, ringing in the ears,

and pulsating sounds in the head. These symptoms are sometimes increased when the patient lies down, and are generally increased when the patient bends the head over so as to prevent the return flow of blood from the brain. It is impossible to diagnosticate passive hyperæmia from active through the symptoms alone, but probably in the former condition the disturbances and symptoms mentioned are less marked; in other words, an active hyperæmia produces more phenomena than passive. In either case examinations of the fundus of the eye and of the tympanum furnish no sure criteria.

Pathology.—Cerebral hyperæmia used to be regarded as nearly synonymous with cerebral neurasthenia. In the writer's opinion, it is secondary to the neurasthenic state; and produced, if it exists in that state, by the impaired vasomotor innervation which is characteristic of neurasthenia. It is not wise, therefore, to make the diagnosis of cerebral hyperæmia often as the primary condition. It is only after traumatisms and sunstroke or after a meningitis that we can speak of the cerebral hyperæmia as being in a certain sense the primary condition to be treated. The statement made by some writers that cerebral hyperæmia underlies certain conditions of acute delirium, of aphasia, of paralysis, and even dementia or insanity can hardly be supported. In many of the cases of cerebral hyperæmia where symptoms are produced, there is undoubtedly a condition of toxæmia which is a contributing factor to many of the symptoms.

Treatment.—The specific treatment of cerebral hyperæmia, when indicated, consists in giving large doses of fluid extract of ergot and bromide of potassium. One or two drachms of the ergot three times a day and fifteen or twenty grains of bromide of potassium may be prescribed. Wet cups to the back of the neck, the cautery in the same region, ice caps, purgatives, quiet and rest, and a careful regulation of the diet and the bowels are all important measures.

Cerebral Anæmia.—This condition, like hyperæmia, may be either acute or chronic.

It occurs among the young ; more often in females than in males. It is seen in early adult life, where it is induced by the various causes producing general anæmia, and again after the climacteric, when it is due to organic changes in the cerebral arteries of the nature of an obliterating endarteritis. Bright's disease and syphilis, exhausting diseases and profuse hemorrhages, and such disorders of digestion and nutrition as lead to general anæmia produce also cerebral anæmia. A potent cause of acute cerebral anæmia is fright.

Symptoms.—The symptoms of acute cerebral anæmia are vertigo, confusion of ideas, nausea, faintness or complete syncope.

In chronic cerebral anæmia the symptoms are mental apathy and a feeling of disinclination to work, tendency to somnolence in the day-time and insomnia at night, mental depression, headaches which are usually frontal or vertical, occasionally some vertigo and tinnitus. There may be spots before the eyes and undue sensitiveness to sounds. In children some very severe symptoms are attributed to cerebral anæmia, but here, as in hyperæmia, it is probable that there are other causes at work, particularly toxic agents or reflex disturbances.

Diagnosis.—A chronic anæmia of the brain can hardly be recognized except through the evidences of a general anæmia. When this is present and there are also symptoms such as have been described, a fairly certain diagnosis can be made. We must look upon cerebral anæmia as being in almost all cases a secondary phenomenon, except in the aged, and then the trouble is due not alone to poverty of the blood, but to the fact that the circulatory apparatus is diseased. It is generally believed that in cerebral anæmia the symptoms somewhat improve by the horizontal position and are made worse by the upright position. It is also asserted that in anæmia the pupils rather tend to be dilated, while in hyperæmia they are contracted.

Treatment.—Treatment should be directed toward enriching the supply of blood and toward improving the general nutrition. It consists, therefore, in the administration of preparations of iron and of such tonics as the mineral acids, strychnine, quinine, and nitroglycerin.

INFLAMMATION OF THE BRAIN—ENCEPHALITIS.

The forms of inflammation of the brain are acute suppurative encephalitis, acute non-suppurative encephalitis, chronic encephalitis, chronic polioencephalitis. Inflammations of the brain are analogous to those of the spinal cord; that is to say, there are acute suppurative inflammations or abscesses, and acute exudative inflammations with or without necrosis, and there are chronic inflammations. The brain differs from the spinal cord, however, in that acute suppurative inflammation is comparatively frequent, while the acute exudative inflammations are extremely rare. Chronic inflammation, also, of the brain is rarer than chronic inflammation of the cord, if we except that type which underlies general paresis.

Acute Suppurative Encephalitis (Abscess of the Brain).

Brain abscess is a suppurative inflammation which affects the parenchymatous and other structures of the organ. It is always a focal disease, but may be single or multiple.

Etiology.—The primary cause of all forms of brain abscess is a microbic infection. The form of microbe, its mode of entrance, and the part of the brain attacked vary greatly. The predisposing causes relate chiefly to age and sex. Brain abscess rarely occurs before the first year or after the fiftieth year of life. It is rather frequent in young people, and occurs on the whole oftenest between the ages of ten and thirty. Males are more often affected than females in the ratio of about three to one. The exciting causes are chiefly disease of the ear and of the cranial bones, injuries, and remote suppurative processes. To this may be added infectious fevers and the presence of tumors. Inflammation of the middle and internal ear is the most common cause of brain abscess. The disease affects the tympanum and mastoid cells, more rarely the labyrinth. It is usually a suppurative process of long standing. Caries of the ethmoid and nasal bones and of the orbital cavity leads to brain abscesses in a considerable proportion of cases. After chronic ear and bone diseases, injuries are the most frequent cause. The injury may be a compound fracture with direct infection from the open wound, or the abscess may be the result of *contrecoup* and may develop in a part of the brain opposite to that which was injured, or the abscess may develop below the point injured, there being apparently healthy tissue between the surface of the brain and the diseased part. These abscesses develop through laceration of brain tissue and subsequent infection of the wound with organisms. The most common remote suppurative processes which are followed by brain abscess are tubercles, inflammation of the lungs, fetid bronchitis, and empyema. Brain abscess may develop, however, from distant points of suppuration on the extremities or in almost any part of the body. Pyæmia may lead to the production of brain abscess. Among the infectious fevers which are complicated with brain abscess are diphtheria, typhoid and typhus fevers, erysipelas, small-pox, the "grippe." The oidium albicans or thrush may also be a cause. Brain tumors sometimes become surrounded by a suppurative encephalitis or may break down with the formation of mixed suppurative and neoplastic tissue. Tubercular tumors are most frequently accompanied by suppurative encephalitis.

Symptoms.—Brain abscesses take sometimes an acute and sometimes a chronic course. In acute cases the symptoms develop rapidly and the disease runs its course in a few days or weeks. The symptoms come under the general head of those of pressure, those of poisoning from the diseased focus, and local symptoms due to irritation or destruction of certain special areas of the brain. The pressure symptoms are those of headache which is often very severe and persistent, vomiting which is quite

frequent though not invariable, vertigo, and a condition of mental dulness which may pass into a delirium, ending finally in coma. Optic neuritis often occurs. The pupils are apt to be irregular, but furnish no definite indications. The pulse is usually slow, ranging from 60 to 70, but it varies a great deal. The temperature is normal or subnormal as a rule, but this also varies, and it may rise several degrees above normal, always running an irregular course. The toxic symptoms are those which we get in septic poisoning; namely, prostration, irregular fever, emaciation, anorexia, and such mental and sensory disturbances as have already been referred to. As a result of local irritation or destruction, there occur convulsions, paralysis, aphasia, and disorders of some of the special senses. Convulsions are not very common. When they occur they are generally of an epileptic character. The paralysis is usually in the form of hemiplegia. The cranial nerves are not often involved, if we except the optic. The urine is said to show a diminution in chlorides and an increase in phosphates. The patient dies finally in coma from exhaustion.

In the chronic form of brain abscess the symptoms may for weeks, months, or years remain practically latent, after the exciting cause has been at work and after the abscess has been established. The patient during this latent stage may suffer from headache, vertigo, mental irritability, and depression; he may at times have a convulsive attack. Occasionally there will be an exacerbation of the disease, at which time he suffers from intense pain, vomiting, perhaps delirium or a convulsion. From this he recovers and continues in a fairly good state of health again. After a variable period, usually of weeks or months, the terminal stage sets in. This terminal stage of the chronic form may assume very much the characters of the acute form already described. In other cases it shows itself by a sudden apoplectic or epileptic seizure or a sudden attack of coma, in which the patient sinks and rapidly dies. These terminal phenomena are due to the fact that the abscess, which has been previously encysted and quiescent, suddenly breaks into a lateral ventricle or through the surface of the brain, or to the fact that a hemorrhage occurs into the abscess.

Complications.—The common complications of brain abscess are a phlebitis of the superior petrosal and lateral sinuses and a meningitis. The phlebitis accompanies abscesses that are caused by disease of the ear. The meningitis may be caused by ear disease, but more frequently accompanies abscesses due to injury. When phlebitis is present there will be found an œdema about the ear and neck and a hardness of the jugular veins. In meningitis there is apt to be more rigidity of the neck, more pain, and there are often cranial-nerve paralyses.

Pathology.—Acute suppurative encephalitis resembles acute suppurative myelitis in the intimate nature of the changes that take place. There is an intense congestion of the parts, which give it a reddened appearance and which used to give to this process the name of *red softening*. This condition, however, is only the initial stage of the suppurative inflammation and does not deserve to be ranked as a special form of inflammatory process. It is possible that in some cases the inflammation may get no further than the stage of red softening. The congestion then gradually disappears, absorption of exudate occurs, and a more or less complete recovery takes place. When the process continues, however, the parts become crowded with leucocytes and infiltrated with inflammatory exudate. The nerve fibres and cells are destroyed, in part mechanically, in part by the poisonous influence of the pyogenic organisms. The nerve cells lose their normal contours, swell up, and disintegrate; the neuroglia cells absorb the broken-down detritus and swell up, forming what are known as granular corpuscles; the leucocytes increase until a purulent mass is formed. The total result is a mixture of broken-down nerve fibres and cells, leucocytes, and granular bodies. Bacteriological tests show the presence of various pyogenic microbes. The abscess thus formed varies in size from one to six or eight centimetres in diameter (two-fifths to three inches). It is generally somewhat round, and if the case is chronic a fibrous wall is formed. It takes from three to four weeks for such a wall to develop (Fig. 178). Brain abscesses are usually single, occasionally there are two or three. In some conditions they are multiple; that is to say, there may be fifteen, twenty, or more. Multiple brain abscesses are always small and are usually due to pyæmic infection.

Location.—Brain abscesses involve the cerebrum oftener than the cerebellum, in the proportion of about four to one (Barr). They occur rather oftener in the right cerebrum. They are very rare in the pons and medulla. The cerebral lobes oftenest affected are the temporal and frontal. In the cerebellum it is the lateral hemispheres that are most frequently attacked. The seat of the abscess has important relations to the cause. Abscesses due to ear disease are almost always either in the temporal lobe or the cerebellum. If the ear disease is in the tympanum, the cerebrum is usually the seat of the abscess. If the disease is in the mastoid cells, the cerebellum is usually the part affected. If the disease is in the labyrinth, the abscess is also more apt to be in the cerebellum. This distribution of the seat of the disease is due to the anatomical relations of the bony parts to the temporal lobe and cerebellum, respectively. Brain abscesses due to injuries are more frequent in the frontal and temporal lobes. What are

known as idiopathic brain abscesses—that is, those which arise without any known cause—are most frequent in the frontal lobes. This is because most such cases are due to an unrecognized affection of the nasal cavities and ethmoid bone. Brain abscesses due to suppurative processes in the lungs and pleura are probably embolic; and as the emboli are carried up into the middle cerebral artery, the brain abscesses having this origin are situated in the field supplied by this artery. In children under ten, in whom

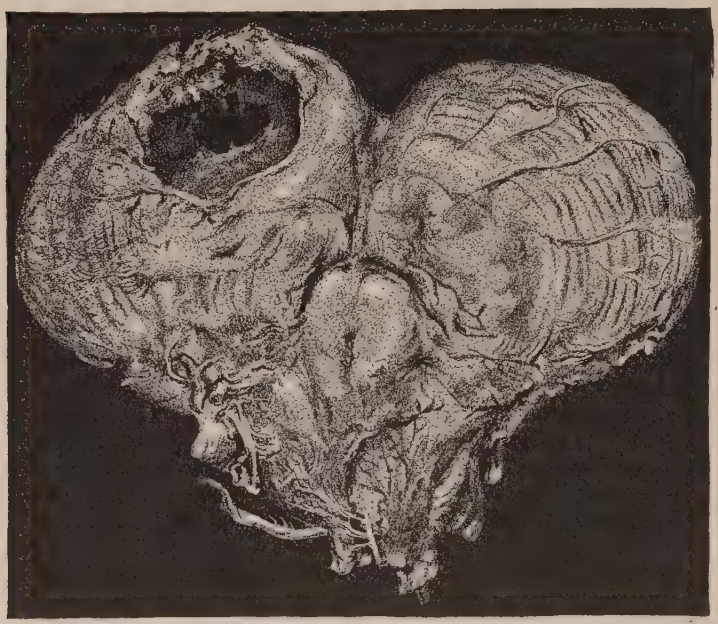


Fig. 178.—Abscess of Cerebellum.

brain abscess is usually due to ear disease, the cerebellum is more apt to be affected.

Course.—Acute abscesses last from five to fourteen days, rarely over thirty days. Traumatic cases run the shortest course. Chronic abscesses may have a latent period of weeks, months, and in rare cases even one or two years. When terminal symptoms come on death occurs in a few days. In a few cases brain abscesses have been spontaneously evacuated through the nose. Aside from this, the termination is always a fatal one unless surgical interference takes place. There is sometimes a recurrence of the abscess after an operation.

Diagnosis.—The diagnosis of brain abscess is based upon the

history of injury, aural or nasal disease, remote suppuration, upon the general symptoms of sepsis, upon the presence of headache, vomiting, slow pulse, normal or subnormal and irregular temperature, a local tenderness of the scalp and rise of tempera-

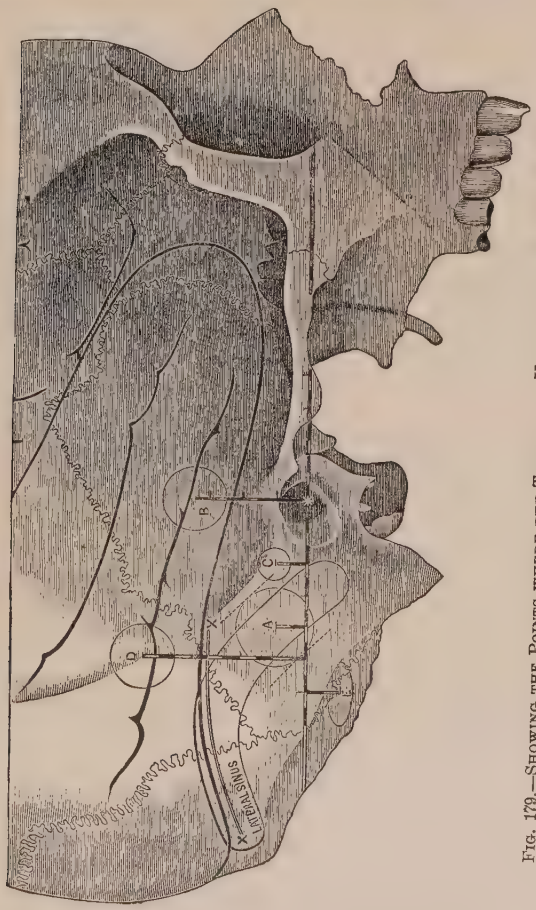


FIG. 179.—SHOWING THE POINTS WHERE THE TREPHINE IS USUALLY APPLIED AND THE RELATIONS OF THE SINUS. The divisions on the lines indicate quarter inches (*Lancet*).

ture over the seat of the lesion, hebetude, delirium, optic neuritis, rapid wasting, and diminution of chlorides in the urine.

The diagnosis of the location of the abscess is based upon the history of its cause, whether from injury, ear disease, emboli from the lungs, or nasal disease; also upon the presence of hemiplegia, local convulsions, tenderness and rise of temperature of a certain area of the scalp. As brain abscesses are apt to affect

latent regions like the temporal and frontal lobes, local diagnosis is usually difficult. The diagnosis must be made from tumors of the brain, meningitis, and phlebitis of the sinuses. The differential points are given under the heads of the diseases mentioned.

The *prognosis* of the disease is absolutely unfavorable unless some surgical interference is resorted to. The few rare cases of spontaneous evacuation of the abscess would not lead to any practical modification of this statement.

Treatment.—The actual treatment of a brain abscess after it has developed is, as already stated, exclusively a surgical one. The successes so far have not been very great, but they have been sufficient to justify operation and to furnish greater hope for the future, when a more exact diagnosis can be made and a wider surgical experience has been obtained. The accompanying figure shows the points to be located in trephining for abscess from ear disease. Something is due to the patient in the way of prevention, especially in cases of persons who have chronic aural or nasal disease with carious processes. These should be carefully watched and treated.

Acute Exudative Encephalitis (Acute Bulbar Myelitis, Polioencephalitis Superior and Inferior, Infantile Polioencephalitis, Primary Encephalitis of Friedmann).

Acute exudative encephalitis is a disease whose pathological existence is probable, but as yet we are not able to establish any definite clinical types associated with the process. An exception to this may be made in the case of acute bulbar myelitis and acute polioencephalitis superior. A third exception is claimed by Strümpell, who affirms the existence of an acute polioencephalitis of children. A form of acute non-suppurative and primary encephalitis is described by Friedmann, and illustrative cases are given; but as yet it is doubtful if we can place this condition in the category of a distinct disease.

Acute bulbar myelitis is a disease which is strictly analogous in course to acute myelitis. The special symptoms simply depend upon the peculiar location of the disease. They consist of an acute glosso-labio-laryngeal palsy, and are referred to under the description of bulbar paralysis.

Acute polioencephalitis superior is a disease in which the nuclei of the nerves supplying the eye muscles are involved; it is a disease analogous to poliomyelitis anterior, and has been described under the head of Ophthalmoplegia.

The acute polioencephalitis of children (Strümpell) is a disease that involves the gray matter of the convexity of one of the

cerebral hemispheres. The disease is supposed to be strictly analogous to an inflammation of the anterior horns of the spinal cord. It attacks children between the first and fourth years of life, with symptoms of fever, delirium, and often of convulsions followed by a hemiplegia. The acute symptoms pass away in a week or two, leaving the child with the hemiplegia which may improve very greatly, but more commonly continues through life. With it later there are developed contractures of the affected side, some atrophy, mental defects, and epileptic convulsions. The real existence of this disease is very doubtful.

Chronic Encephalitis.—Under the older views of inflammation there were described as chronic cerebritis such lesions as sclerosis and cerebral atrophy. These processes we now regard as being simply reparative and as the sequelæ of acute destructive or inflammatory processes. They will be described, therefore, under such heads. The only form of chronic cerebritis for which there is a recognized clinical prototype is the chronic cortical cerebritis, which is the basis of general paralysis or paralytic dementia. Even this process, however, is still regarded by many as essentially a degenerative one and not inflammatory. As the symptoms which result from it are those of insanity, the disease will not be described in this work.

THE APOPLEXIES.

Apoplexy is a condition characterized by a sudden paralysis and shock, usually with loss of consciousness, and due to the breaking or blocking up of a blood-vessel in the brain.

Apoplexy is a general term. Its particular forms are:

1. Intracranial hemorrhage, from rupture of blood-vessel.
2. Acute cerebral softening, from embolism or thrombosis.

Since both embolism and thrombosis cause a rapid softening in the area supplied by the obstructed vessel, the term acute softening covers both disorders.

Apoplexy from Intracranial Hemorrhage (Cerebral Hemorrhage, Hemiplegia).

Forms.—There are four groups of blood-vessels in the brain, those of the dura mater, those of the pia mater, and those supplying the basal ganglia and white matter. Besides this, we may consider the pons, medulla, and cerebellum which are supplied chiefly by branches of the vertebrals as a separate group, subject to somewhat different mechanical conditions. Corresponding to this we have:

1. Dural or pachymeningeal hemorrhages.
2. Pial or subarachnoid hemorrhages.

3. *Central hemorrhages.*

4. Hemorrhages in the medulla, pons, and cerebellum.

It is the central hemorrhages (No. 3) due to rupture of the blood-vessels going to the great basal ganglia, internal capsule, and white matter that cause the great majority of cerebral hemorrhages seen by the physician. It is this class that I have particularly in mind in the following description.

Etiology.—At the time of birth and during infancy there is a slight tendency to intracranial hemorrhage owing to the accidents and injuries of labor. After this period the liability is very small, but slowly increases up to the age of forty, when predisposition specially begins. Four-fifths of all cases occur after forty, and the tendency to hemorrhages increases in each decade up to eighty, when it diminishes absolutely and relatively.* Males are slightly more predisposed than females (five to four). Rather more cases occur in cold weather, at high altitudes, in the temperate zone, and among civilized races. Heredity has an undoubted, though not great influence in predisposing to cerebral arterial disease. Infective fevers and marasmic states are predisposing causes. Chronic kidney disease is present in one-third of the cases. Chronic alcoholism, syphilis, and gout are powerful predisposing causes. Rheumatism is less important. Heart disease, fatty and atheromatous arteries, arteritis, and miliary aneurisms may be regarded as more than simply predisposing—they are determining causes. Leucocythæmia, scurvy, and purpura are conditions which also particularly tend to cause hemorrhage. The so-called apoplectic habit—short thick neck and high shoulders and florid face—has really some importance in the better classes. Congenital anomalies, such as a narrow thoracic aorta or inherited deficiency in the strength of the walls of the blood-vessels, also play a part. Any sudden physical exertion, such as straining at stool, the excitement at coitus or of a passion, eating a large meal and drinking a great deal of fluid, especially alcohol, taking a cold bath, all may lead to rupture of an artery. Often the vessel breaks during sleep, owing perhaps to the contracted condition and consequent higher pressure in the arteries, and to the fact that on account of the horizontal position there is more resistance to the return of the blood from the brain.

The *symptoms* are the prodromal, those of the attack and acute stage, and those of the chronic stage.

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* Among 53 cases collected by me at Bellevue Hospital, the ages were 10 to 20, 4; 21 to 30, 6; 31 to 40, 10; 41 to 50, 11; 51 to 60, 7; 61 to 70, 10; 71 to 80, 5. The right side was affected in 23 cases; left in 25 cases. The location was: Pachymeningeal, 7; pial and cortical, 8; ventricular, 23; corpus striatum and vicinity, 7; optic thalamus, 2; corpora quadrigemina, 1; pons, 1; cerebellum, 3.

Prodromal symptoms are rare except in syphilitic cases. When present the patient suffers from dizziness, numbness of the hand and foot on one side, and a failure of memory for words. He may have "full" feelings or even pain in the head and bad dreams at night. Nose-bleed and irregular heart action sometimes occur. The attack always comes on suddenly and may be accompanied (1) with convulsions and coma, (2) with coma alone, or (3) without loss of consciousness.

1. Initial convulsions are rare and generally mean a meningeal hemorrhage. When present they are unilateral or partial, as a rule, but may be general. 2. The common mode of onset is with coma. The patient, without warning, suddenly becomes dizzy, loses consciousness, and falls. The face is flushed, the pulse hard and rather slow, the breathing is labored and stertorous, the cheek on one side puffs out with each expiration, the eyes are partly closed, the eyeballs fixed or deviated to the paralyzed side, the pupils are contracted and rigid, the skin is bathed in sweat, the limbs are relaxed, but some evidence of hemiplegia is present; the urine may be retained or it and the fæces involuntarily evacuated. The urine is usually of rather high specific gravity and often contains albumin, even when there is no renal disease. The temperature in severe cases may fall below normal during the first twelve hours, even to 96° F., but this is not the rule. It is the rule, however, for the temperature in a few hours to be 1° or 2° higher on the paralyzed than on the sound side. If the case is rapidly fatal coma continues, respiration often assumes a Cheyne-Stokes character, the pulse becomes faster, the temperature gradually rises, and usually reaches 102° or 103° F., until just before death, when it may sink again. Swallowing and speech become difficult, hypostatic pneumonia sets in, and the patient dies in from two to four days. In slower fatal cases the patient regains consciousness partially and then enters a condition of stupor or mild delirium. He is restless and suffers from headache. The temperature may continue normal for a time, but is usually higher on the affected side. At the end of two or three weeks it rises higher, pneumonia develops, the patient becomes unconscious and finally dies. In the favorable cases, which constitute the majority, coma, if present, gradually passes away in from one to six hours, leaving the patient's mind somewhat weak and confused and his speech disturbed, or more rarely the intelligence may not be at all disturbed. During the first few days or weeks after the attack the physician finds that the prominent symptom is the hemiplegia. This affects the arm and leg most and the face least. Only the lower two branches of the facial nerve are involved, and the patient can shut the eyes. The tongue, if protruded, turns to the paralyzed side; the uvula is turned in various

ways and its position is of no significance. There is often some evidence of cutaneous anæsthesia of the paralyzed side, and less often hemianopsia and disturbances of hearing occur. In right-sided hemiplegia the patient, after recovering consciousness, is often unable to talk or to understand what is said. Examination shows that he has a motor or sensory aphasia (*vide* Aphasia).

The deviation of the eyes and head to one side usually disappears in a day or two. Occasionally there is a temporary ptosis. The pupils at first are contracted, that on the paralyzed side the more so; this condition disappears with returning consciousness.

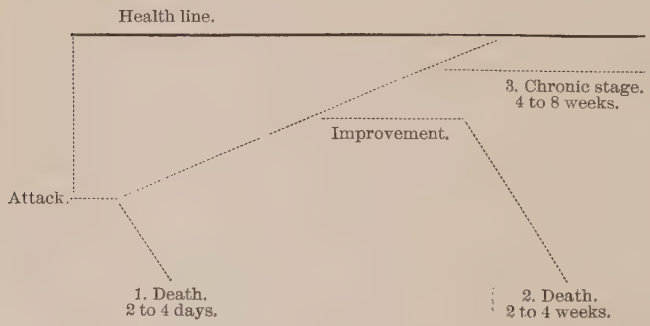
The paralysis of the arm and leg is flaccid at first, and the limb falls heavily when lifted; the reflexes are lessened or abolished.

Sometimes, however, rigidity sets in at once. This symptom occurs when blood has broken into the ventricles, and also in some meningeal hemorrhages. The skin reflexes are abolished or nearly so on the affected side.

The usual course of the temperature is for it to rise on the second and third day to 100° F. or 102° F., being $\frac{1}{2}$° to 1° F. higher on the paralyzed side. In a few days it gradually falls, so that by the eighth to the tenth day it is normal.

If the temperature continues to rise after the fourth or fifth day, it is a sign of an extension or inflammatory reaction of the hemorrhage. Hence the thermometer furnishes a very important criterion of the seriousness of the case.

The varying course of the apoplexy is shown in the following diagram:



The Chronic Stage, Hemiplegia.—At the end of a month, if fever and symptoms of cerebral irritation have subsided, the chronic stage may be said to begin. The hemiplegia has improved, the patient can move the leg and arm a little, sensory symptoms

have lessened, the mind is clear, headache has disappeared. Improvement continues, though more slowly, for several months or even one or two years. During this time the patient is "a hemiplegic."

The hemiplegia affects the arm more than the leg and the face least of all. The distal segments of the limbs, the feet and hands, are affected more than those near the trunk. The muscles that act bilaterally, such as those of respiration, phonation, and facial expression, are but slightly involved. The paralysis is not strictly a hemiplegia, for the muscles on the sound side are somewhat weakened, as tests will show. In severe cases, especially in old people, even the visceral muscles, especially those of the bladder, are weakened. At the onset of the attack there is sometimes a temporary "initial" rigidity of the muscles on the paralyzed side, or an "early" rigidity may develop in one or two days. There always develops at about the beginning of the second week a "late" rigidity. This, which at first is slight, gradually increases, and finally contractures affect the paralyzed limbs. The superficial reflexes, which at first were absent, reappear; the tendon reflexes become much exaggerated, and clonus can be obtained in the leg and arm. The sound side shares to a small extent in these conditions. The contractures affect the extensors of the foot more than the flexors, and bring the toe down and the heel up. The leg is held nearly extended, and the limb in walking is swung around, the toe scraping the ground. The shoulder is adducted, the forearm flexed, and the fingers tightly shut into the palm by the overaction of the flexors. The facial muscles show a slight contraction and drawing to the affected side. The muscles on the paralyzed side do not waste. In infantile hemiplegia, however, the affected limbs grow less than those on the sound side.

The paralyzed limbs may be the seat of peculiar disorders of movement. These consist of

Associated movements.

Tremor.

Ataxia.

Choreic movements.

Continuous or athetoid movements.

Spastic movements and cramps.

Such movements, aside from those that are spastic, are rarely seen in the hemiplegia of adults.

The electrical irritability may be at first sightly increased and diminished, but the change is slight in amount and never reaches the degenerative stage.

Hemianæsthesia, if present at first, disappears to a great extent, leaving only residua about the feet and hands. Paræsthe-

siæ are common. In rare cases the patient suffers great pain in the arm and leg. This pain is generally of a burning character and very obstinate and distressing. Cramping pains in the legs and arms are common in the severer cases.

During the first five weeks after the onset joint inflammations and bed-sores may attack the affected side. The temperature of the hemiplegic side is usually a very little higher than that of the sound side.

Vasomotor disturbances, sweating, skin eruptions, and increased growth of hair are some of the rarer symptoms.

The mental condition is more or less affected. The patient becomes irritable, cries easily, and is in general more emotional. The memory is impaired, and the power of concentrating the attention and carrying on work is less. Sometimes a progressive mental deterioration sets in or insanity develops. The mental disturbance is greater in old people and depends somewhat on the size of the hemorrhage. Those forms which produce serious aphasia especially limit and lessen mental activity.

Pathology and Morbid Anatomy.—Spontaneons intracranial hemorrhage is always due to the presence of diseased blood-vessels in the brain. This diseased condition consists of

1. A degenerative arteritis which results in producing small aneurisms. 2. A fatty degeneration of the vessel walls. 3. Besides this, in most cases the larger blood-vessels are atheromatous.

1. The arteritis produces small or miliary aneurisms which affect only the smaller arteries, especially those of the central group. They may be fusiform or sacculated in shape; they range in size from one-fifth to one millimetre ($\frac{1}{100}$ to $\frac{1}{25}$ in.) in diameter. They are usually not very numerous, but there may be as many as a hundred in the brain. They are the results, not of inflammation, but of a degeneration which affects first an area in the internal coat; this causes local weakness and consequent dilatation; secondarily there is a periarteritis. These aneurisms occur almost exclusively during the degenerative period of life.

2. Fatty degeneration of the walls of the small cerebral arteries occurs in purpura, scurvy, leucocythæmia, marasmic conditions, and post-infective states, especially in early life, and is the common cause of hemorrhage at that time.

3. Atheroma affects the larger vessels only. It is indirectly a cause of hemorrhage by lessening the elasticity of the vessel wall. Atheroma is present in one-eighth to one-fifth the cases. Hypertrophy of the heart is a factor in causing hemorrhage, and such hypertrophy exists in about forty per cent of cases. Emboli lodged in the cerebral arteries may cause hemorrhage by suddenly stopping the arterial circulation and raising the blood pressure. Hemorrhages are found by far the oftenest (twenty per

cent) in the caudate and lenticular nuclei and adjacent parts. The lenticular and lenticulo-striate branches of the middle cerebral are oftenest affected; next the branches of the anterior cerebral to the caudate nucleus and the ventriculo-optic branches of the middle cerebral. The branches of the posterior cerebral break more rarely. The parts affected in hemorrhage, in order of frequency, are about as follows:

Caudate and lenticular nuclei.
Meninges and cortex.
Centrum ovale.
Optic thalamus.
Pons, cerebellum, medulla.

Cortex hemorrhages are generally small and may be subarachnoid or may break through into the arachnoid cavity. Ventricular hemorrhages are almost always secondary to a rupture into the neighborhood of the basal ganglia. Pons hemorrhages occur usually in the median line. Cerebellar hemorrhages are oftenest due to rupture of the superior cerebellar artery. They usually cleave their way externally and break into the fourth ventricle. Dural hemorrhages are due to rupture of the meningeal veins and arteries and of the vessels in newly organized clots. They lie in the arachnoid cavity and flatten the convolutions.

The reparative changes after a hemorrhage take the following course: 1st. Coagulation of the blood, which in a few days begins to soften and become absorbed. 2d. Formation of a fibrinous wall about the clot. This occurs from the seventh to the ninth day. 3d. Formation of a cyst with transparent fluid contents, and perhaps fibrous trabeculæ running through it, twentieth to thirtieth day. 4th. Contraction of the cyst wall, which begins by the fortieth day. 5th. Secondary degenerations begin from the tenth to the fourteenth day.

Physiology.—The blood pressure of the cerebral arteries is equal to about 150 mm. of mercury. The resistance or support furnished by the surrounding tissue is equal to about 10 mm. of mercury. Hence there is a special liability to rupture of intracranial vessels. The middle cerebrals are most often affected, because they are in the most direct line from the heart and are nearest to that organ. The pressure lessens as the arteries subdivide and get farther away from the heart (Mendel).

The *diagnosis* must be made from alcoholic coma, uræmic coma, opium coma, epilepsy and hysteria, acute softening from embolism and thrombosis.

From alcoholic coma the diagnosis is made by the odor of the breath, the incomplete coma, the equal pupils, the absence of low or unequal temperature.

From uræmic coma by the absence of albumin and casts in the

urine, though their presence does not surely indicate uræmia; by the unequal pupils, the temperature, the absence of hemiplegia, and of the physiognomy peculiar to cases of chronic Bright's disease.

From opium-poisoning by the history, the stomach contents, the presence of equal and contracted pupils, the slow respirations, the temperature, and the absence of paralysis.

From epilepsy by the history of the onset with epileptic cry, the dilated and equal pupils, the biting of the tongue, the absence of hemiplegia, the rather rapid return of consciousness.

Hysterical attacks present little semblance to that of apoplexy; hysterical hemiplegia is characterized by its flaccidity, by its not involving the face, and by the presence of the anæsthesia and other hysterical stigmata.

In embolic softening the earlier age of the patient, the presence of decided valvular heart disease, the parturient condition, the slighter degree and shorter duration of coma, the absence of serious disturbance of temperature, the onset first of paralysis and then of convulsive movements and coma—all lead to a presumption in favor of embolism.

The presence, on the other hand, of a congested face, tense pulse, and throbbing carotids favors the existence of a hemorrhage.

From thrombotic softening diagnosis is more difficult. The occurrence of prodromata, consisting of slight seizures quickly recovered from, the slighter degree of coma, the advanced age, hard atheromatous arteries, evidence of anæmia and asthenia, weak or fatty heart, the absence of stertorous respiration, flushed face, and unequal temperature not much lowered or raised, the slight pupillary disturbance, and absence of convulsions point to thrombotic softening. Evidence of a lesion in the pons or cerebellum suggests hemorrhage, while evidence of lesion in the medulla points almost surely to softening.

The chances in any case between the ages of thirty and fifty, if there is no heart disease, are six to one in favor of hemorrhage.

Prognosis.—The majority of cases get over the first attack. They are very liable to have another within one to five years. The minority recover from this. Few survive a third attack. The prognosis of the attack itself depends on the severity of the coma and paralysis, the disturbance of temperature and of respiration, the evidence of rupture into the ventricles, the development of decubitus, the continuance of loss of control over the bowels and bladder.

If profound coma continues four days there is little hope; if fever develops and continues steadily, or if there is initial subnormal temperature, the prognosis is grave.

If the patient passes the first week with little or no fever and consciousness has returned, the prognosis is good.

The presence of renal disease and of alcoholism is bad. Development of slight delirium which continues is unfavorable.

Cerebellar and pons hemorrhages are very fatal, meningeal slightly less so.

The prognosis of the chronic stage has been given under symptoms.

Improvement continues rather rapidly for three months, then very slowly. Improvement may continue for one or two years. Complete recovery is very rare. The great danger after middle age is recurrence of the attack.

Treatment of the Attack.—The patient should be laid in a horizontal position and kept quiet. Ice should be applied to the head and hot bottles at the feet. The feet and legs should be swathed in clothes wrung out in hot water containing mustard, a cupful to a pail of water. A laxative should be given, either one or two drops of croton oil or a quarter of a grain of elaterium. If there is evidence of intense cerebral congestion, the pulse being very full and hard and the heart beating strongly, bleeding eight to ten ounces is justifiable. Ordinarily it is better to give a drop of tincture of aconite every twenty minutes for two or three hours. Pressure on the carotid of the sound side, and even ligature of it, has been recommended, but there is no experience yet to justify it. Administration of bromide of sodium and enemata of ergot have been advised, but are of doubtful value. After the first twelve hours treatment must be symptomatic. Should delirium and other evidence of mental irritation appear, large blisters must be applied at the back of the neck and an elaterium purge given if the patient is not too weak. The use of iodide of potassium or mercury is not indicated unless the case is distinctly syphilitic. The passage of a galvanic current through the brain cannot possibly do any good. Great care should now be taken that the patient does not develop pneumonia. The mouth and pharynx should be cleansed antiseptically, and the patient should not be allowed to remain in one position. If there is sufficient evidence of a meningeal or cortical clot, trephining should be seriously considered.

At the end of three or four weeks the faradic battery may be used carefully on the affected limbs. A séance of fifteen minutes daily for four to six weeks should be given, then treatment should be suspended for a fortnight, to be begun again and kept up systematically for a year if need be. Massage may be alternated with the electricity. When contractures develop the stabile galvanic current may be tried, though it does little good. Static sparks, however, are helpful: lukewarm baths should be tried;

and measures used to produce hyperextension of the affected parts.

Internally during this time the patient is to be given courses of iodide of potassium, tonics, and laxatives if needed. The patient should be made to live a quiet life, preferably in a warm, equable climate. The kidneys should be kept active and arterial tension low. For these purposes nitroglycerin should be given and at times small doses of chloral, and the diet should be simple and rather non-nitrogenous. Strychnine in very small doses (gr. $\frac{1}{100}$) sometimes helps the contractures; so also do the bromides and physostigma.

Acute Softening of the Brain (Embolism, Thrombosis).

Acute softening is a condition caused by the plugging of a blood-vessel with an embolus or thrombus, and is characterized by a sudden apoplectic seizure; the symptoms eventually running a course like that of cerebral hemorrhage.

Etiology.—Embolism occurs rather more often in women, thrombosis in men. Embolism is rare in children; it occurs oftenest between the ages of twenty and fifty, thrombosis between the ages of fifty and seventy. The most important predisposing factors in embolism are acute or recurrent endocarditis, infectious fevers, profound anæmia, pregnancy, and blood dyscrasiæ; in thrombosis, syphilitic, lead, or gouty arteritis, fatty heart, and blood dyscrasiæ. The same causes which lead to the arterial disease which produces cerebral hemorrhage also predispose to thrombosis; though in the latter condition atheroma plays the important part.

Symptoms.—In embolism there are rarely any premonitory symptoms; the onset is sudden; it may begin with some convulsive twitchings, then follow hemiplegia and temporary loss of consciousness. Coma, however, is rarer than in hemorrhage, and if present is usually shorter. There is rarely vomiting, nor do we find the hard, pulsating arteries, flushed face, and severely stertorous breathing. The initial temperature changes are slight, but in a few days fever may develop.

In thrombosis premonitory symptoms are frequent. In syphilitic cases there are headaches and cranial nerve palsies. In other cases vertigo, temporary aphasia, transient hemiplegia, numbness of the hand and foot, and drowsiness may be present. The onset is more gradual; the hemiplegia slowly develops, taking several hours, perhaps, for its completion; meanwhile the patient gradually becomes comatose. The attack sometimes is rather sudden, with no loss of consciousness, and it may occur in sleep. The temperature often has a

slight initial fall, followed by a rise, just as in hemorrhage. In both embolism and thrombosis the hemiplegia tends to improve very much in a few days or weeks unless the vessel obliterated is a large one. Embolism is rather more apt to affect the left side of the brain, though the difference is not great. The middle cerebrals are most frequently affected (seventeen out of twenty-seven cases). Softenings affect the vertebrals, basilar, and posterior cerebral arteries more often relatively than do hemorrhages; then the initial symptoms may not present the character of hemiplegia, but of a bulbar paralysis. Acute softening may kill within twenty-four hours, but, as a rule, the patient survives the onset, and if he dies it is not for several weeks. After the acute stage is over the patient passes into the chronic stage, which resembles in nearly all respects that of hemorrhage. After an acute softening, however, it is believed that there are more spastic symptoms and a greater tendency to mobile spasm. In embolism, owing to the youth and freedom from arterial disease, the mind is less affected; while in thrombosis the contrary is the case.

Pathology.—The embolic plug cuts off the blood supply from a certain area of brain tissue. In twenty-four hours this begins to soften. If the area is in the cortex it becomes red (red softening); if in the white and less vascular part, it is usually white with a few red punctate spots. The red softening gradually becomes yellow (yellow softening). The dead tissue softens and is absorbed, leaving a cicatrix or cyst. If the embolus contains infective microbes there may be a local encephalitis and abscess.

In thrombosis there are usually evidences of extensive atheroma or of syphilitic arteritis. In those instances in which the thrombosis is caused by the blood state and a weak heart, little arterial change occurs. Atheroma affects chiefly the internal carotids and the large arteries at the base, viz., the middle, anterior, and posterior cerebrals and the basilar and vertebrals. Thrombosis with apoplexy occurs oftenest in the corpora striata and optic thalamus, next in the pons and medulla. Embolism almost always affects the great basal ganglia or some cortical branch of the anterior and middle cerebrals. The secondary changes after thrombosis resemble those after embolism; a thrombus, however, may lead to supplementary embolism through breaking off of a clot, and both conditions may cause a complicating cerebral hemorrhage.

The Diagnosis.—The important points have been gone over under the head of hemorrhage. They may be tabulated in part here.

Hemorrhage.

Age, thirty to fifty.

Hereditary history of arterial disease.

Sudden onset, with coma and paralysis occurring together, the coma deepening.

Initial and early rigidity.

Very unequal pupils.

Stertorous breathing and hard, rather slow pulse.

Peculiar alternating conjugate deviation.

Early rigidity.

Peculiar disturbances of temperature, as described.

Acute Softening.

Earlier or later age.

History of syphilis.

Premonitory symptoms and more gradual onset, more transitory coma, or absence of coma.

Initial convulsive movements.

Presence of weak heart or endocarditis.

Slight hemiplegia with anæsthesia.

The puerperal state.

Embolism is distinguished by the age, the presence of endocarditis, of the puerperium, or infective fevers, and by the sudden onset, with perhaps some convulsive movements. Thrombosis occurs oftener in the aged, and there are prodromata—a slower onset and evidence of arterial disease and a weak heart.

The *prognosis* as regards the attack is somewhat better than in hemorrhage as a rule. In embolism it is good as regards recurrence; in thrombosis, bad. The mental condition is better in embolism; usually worse in thrombosis. The recovery from attacks is more complete in acute softening. After the chronic stage is reached, however, the prognosis is about the same in all forms.

The *treatment* of the attacks consists essentially in rest and such attention to the bowels, kidneys, and heart as may be indicated. In thrombosis it may be important to give heart stimulants and arterial depressants, and for this purpose I advise the use of alcohol, digitalis, or strophanthus with nitroglycerin. Iodide of potassium and mercury ought to be given if there is the slightest suspicion of syphilis. Later it is well to give courses of the iodides and mercury and of strophanthus, nitroglycerin, strychnine, and such tonics as may be indicated. The symptomatic treatment of the chronic stage is the same as in hemorrhage.

Cerebral Palsies of Children—Hemiplegia, Double Hemiplegia or Diplegia, and Paraplegia.

The brain palsies of early life show themselves in the form of 1, hemiplegias; 2, diplegias or double hemiplegias, in which both sides of the body are involved; and, 3, paraplegias, in which the lower limbs are chiefly or entirely involved. In these palsies, as in the same troubles of adult life, the loss of motor power is always accompanied with a rigidity and with some contractures and exaggeration of reflexes, in this respect distinguishing these paralyses from those of spinal origin. The seat of lesion in these cases is in the hemispheres of the brain, and it is the upper motor segment which is involved; that is to say, that part of the motor tract which extends from the brain cortex down to the spinal cord as far as the anterior horns. The brain palsies of children are therefore disorders of the cortico-spinal segment, while the spinal palsies of children are disorders of the neuro-spinal segment.

Etiology.—The disease occurs rather oftener in males than in females, though the difference is slight. The vast majority occur in the first three years of life; about one-third of them are congenital. Injuries to the mother during the time of pregnancy, possibly diseases and emotional disturbances at this time, are factors in producing the congenital cases. Those cases that occur at the time of birth are due to tedious labor, the use of forceps, and other injuries at the time of parturition. After birth, the causes are those which lead to the production of intracranial hemorrhages, embolism, and thrombosis; these being injuries and the infectious fevers. Of the latter, pneumonia, whooping-cough, measles, and scarlet fever are the most prominent. Syphilis is a rare cause; cerebro-spinal meningitis and epileptic convulsions are also occasional causes.

Symptomatology.—The disorder in about one-fourth of the cases begins with a convulsion, which may be unilateral, but is usually general in character, and may last for several hours. At the same time a febrile process develops, and this continues for several days. When these acute symptoms have subsided, or before this, it is noticed that the child is paralyzed upon one side, the paralysis involving the arm, leg, and face as in adult hemiplegia, or perhaps involving both sides. This paralysis undergoes gradual improvement, the face recovering earliest and most, the leg next, and the arm least. As the child develops it is found that the paralyzed side fails to grow as fast as the other, and there may be half an inch to one or two inches of shortening in the arm or leg. The circumference of the limbs is less, the sur-

face somewhat colder, and some vasomotor disturbance may be present. With the progress of the case a rigidity of the affected limbs develops: the heel becomes drawn up, so that there is talipes equino-varus or equino-valgus. The flexors of the forearm and of the wrist and fingers contract, as do also the adductors of the thighs. In general it will be found that there is a contraction of the flexors and adductors of the affected limbs. With this rigidity and the contractures there is an exaggeration of reflexes and clonus in most cases. In the disordered limbs the peculiar mobile spasms develop. These consist of athetoid, choreic, and ataxic movements, also sometimes tremors and associated

FIG. 180.—POST-HEMIPLEGIC ATHETOSIS. BEGINNING IN CHILDHOOD.

movements. The choreic and athetoid movements are the most common (Fig. 180).

Along with the development of these symptoms it is noticed that there are disturbances in the mental condition of the child. It is usually backward in development, this backwardness ranging from simply a feeble-mindedness to complete idiocy. Taking all cases, there is about an equal division between feeble-mindedness, imbecility, and idiocy (Sachs). Perhaps a little over one-fourth of the cases have a fair intelligence. There is usually slowness in learning to talk, and in a small proportion of cases there is a decided aphasia. Such condition is rather more frequent with right hemiplegia than with left hemiplegia, though the rule is not an absolute one. In connection with the mental defect there may develop many of the peculiar moral traits as-

sociated with idiocy and low degrees of intelligence. Epilepsy very frequently complicates the disease; nearly one-half of the cases suffer from this trouble. This epilepsy is in most cases general in character; in a few cases it takes the Jacksonian type, in a small number *petit mal* alone is noted. Examination of this class of sufferers reveals, aside from the paralyses described, various evidences of defective development. These are known as stigmata of degeneration; though they cannot be classed strictly among such, since they are acquired stigmata in most cases, rather than marks which are the result of primary deficiency in development. These stigmata consist of a microcephalic or a macrocephalic skull, cranial and facial asymmetry, prognathism, imperfectly developed teeth, and a high palatal arch. It has been found that, as a rule, in cases of cerebral hemiplegias of childhood the patient eventually has a slight flattening of the skull on the side of the lesion (Fisher and Peterson). Finally, in a few cases there may be found defects in the special senses, such as imperfect hearing, deafness, deaf-mutism, and defects in vision such as hemianopsia, and perhaps imperfections in smell and taste. Anæsthesia is never observed.

The symptoms in the cerebral palsies of children having passed the acute stage and having become somewhat ameliorated, enter into a chronic stage. This chronic stage begins within a few months after birth or after the onset of the disease. No great change occurs in the paralyses as the child grows older until he reaches the time of puberty, though there is a slight improvement in most cases. After the time of puberty, if the mental condition of the child is good, the physical symptoms are apt to improve considerably.

Morbid Anatomy.—The primary changes that lead to the cerebral palsies of children are 1st, simple agenesis or lack of brain development, producing localized atrophy of the cerebrum and the condition known as *porencephalus*. True porencephalus is a condition in which, owing to a congenital defect in nutrition, a cavity or depression exists in the cerebral hemispheres, this cavity reaching generally into the lateral ventricle. True porencephalus is found in about one-fourth of the cases, though no definite statistics can be given, owing to the different interpretations given to this term; 2d, hemorrhage, which is probably the most frequent of the single causes; 3d, embolism; 4th, thrombosis; 5th, meningoencephalitis and perhaps polioencephalitis; 6th, a diffuse cortical sclerosis. Many other terms are used to describe the pathological conditions found at the basis of the brain palsies of children; but the principal causes of all are undoubtedly as has been described—hemorrhages, embolism and thrombosis, and a defective development or agenesis. It is prob-

able that in the hemiplegics the original lesion is generally a central hemorrhage, less often a meningeal hemorrhage. After this, probably the most frequent condition is a porencephalus from some intrauterine accident which may have been defective nutrition causing anæmia and softening, or hemorrhage or thrombosis. Polioencephalitis or inflammation of the cortex of the brain of the kind similar to poliomyelitis is alleged to be a cause in some cases by Strümpell, but this has not yet been proven. In double hemiplegias or diplegias of children the cause is in the

Fig. 181.—Atrophied Brain with Sclerosis and a Cyst, from a Case of Infantile Cerebral Hemiplegia.

vast majority of cases a meningeal hemorrhage due to some injury or disturbance at the time of labor. In other cases of diplegia the lesion is a double porencephalus, which may be either the result of an intrauterine hemorrhage or simply a defective development. In the paraplegias the lesion is probably very much the same as in the diplegias, that is to say, either a meningeal hemorrhage or a true porencephalus. Occasionally a diffuse sclerosis has been found in these cases. Not infrequently, as the result of hemorrhages, there develop cysts which fill up the atrophied areas of the brain (Fig. 181). It is difficult to present accurately and definitely the relations between the pathological

change and the clinical result, but it may be shown with some degree of correctness in the following table:

Original Lesion.	Later Pathological Condition.	Clinical Result.
Hemorrhage. Embolism. Thrombosis. Agenesis.	Atrophy. Lobar sclerosis. Cysts. Porencephaly. Microcephaly.	Hemiplegia. Diplegia. Paraplegia. Sensory defects. Mental defects. Epilepsy, etc.

Diplegia or Birth Palsies.—That form of the brain palsies of childhood characterized by double hemiplegias or diplegias has certain special characters which lead to its being often classed apart. These diplegias in almost all cases are congenital and are due either to injuries at the time of birth or to some disorders of intrauterine life. There may be convulsions or a prolonged state of asphyxia at the time of birth. After recovery from this no special trouble is noticed with the child by the mother for some weeks or months, when it will be found that it does not use its arms or legs. Other convulsions develop, and eventually the features of a double hemiplegia with mental impairment and epilepsy are observed (Figs. 182, 183). In these cases the mental defect is much more decided than in the hemiplegias; indeed, few of these cases ever show any good amount of intelligence. Epilepsy is extremely common. The anatomical lesion in the cases is, as already stated, either a meningeal hemorrhage which has pressed upon and injured the cortical motor areas in each hemisphere or it is a congenital porencephalic defect.

Spastic Cerebral Paraplegia.—In a few cases the brain lesion is such that there results very little disturbance to the arms or face, and the paralysis is almost entirely confined to the lower extremities. Such cases were once described as spastic paraplegias of childhood and thought to be due to lesions in the spinal cord. There is little doubt, however, now that the primary trouble is in all cases a bilateral lesion of the cerebral hemispheres with a descending degeneration affecting a portion of the lateral columns of the cord. In these cases there are rigidity and contractures, club foot, exaggerated reflexes and spasms of the flexors and the adductors, so that the child's legs cross each other and interfere in its feeble attempts at walking. Spastic paraplegia is usually, like diplegia, a birth palsy and due to the same causes. It seems also to be associated with defective intelligence to about the same degree as with diplegias. Epilepsy also is common with this type of the disease.

Diagnosis.—The clinical diagnosis of cerebral palsies is to be made from the spinal palsies. The latter are distinguished by the

fact that in the paralyses of spinal origin there is no rigidity or exaggeration of reflexes, and there are electrical degenerative reactions of the muscles and decided wasting of the limbs with shortening. The mode of onset in cerebral palsies and their distribution in the form of hemiplegias in which the face is involved also indicate the seat of the lesion. The pathological diagnosis

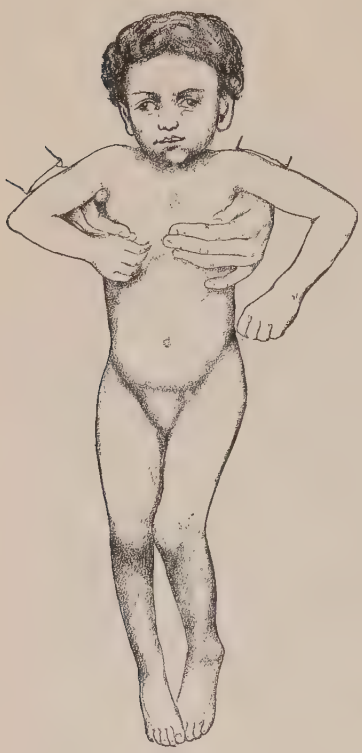


FIG. 182.

FIG. 183.

FIGS. 182 AND 183.—CEREBRAL DIPLEGIA WITH CONTRACTURES AND TALIPES. Fig. 182 standing; Fig. 183 suspended by arms.

is by no means an easy one. Cerebral palsies occurring at the time of birth and accompanied at that time with general convulsions or asphyxia may be considered to be due to meningeal hemorrhage, especially if the delivery of the child has been brought about by the use of forceps or if the labor has been long and tedious. Diplegias and paraplegias which are congenital are probably due to true porencephalus, provided there was no diffi-

culty at the time of labor and no convulsions or other serious phenomena after it. Cerebral palsies occurring after birth in the first, second, or third year of life are apt to be due to hemorrhage, and less often to embolism, or thrombosis. Hemiplegias developing after infectious fevers are likely to be due to hemorrhage. In diagnosticating the pathological lesion in such cases it must always be remembered that the hemorrhage is much more frequent than embolism, and that thrombosis as a factor has not yet been very clearly established.

Course and Prognosis.—In all types of the disease the course is chronic and perfect cure is hardly possible, although in the slighter forms of hemiplegia nearly all traces of the paralysis may be absent. In the hemiplegic form the patient often reaches adult life, and if his intelligence is not defective and he has no epilepsy, the motor trouble improves a great deal and he may live a long and useful life. If epilepsy and mental defect are present, there ensues eventually a further mental deterioration, and such cases rarely live much beyond the period of adolescence, or if they do they pass into the asylums for the idiotic and epileptic. The diplegic and paraplegic cases have a much worse prognosis both as to duration of life and as to improvement in symptoms. The degree of intelligence and the absence of epilepsy are the two factors which measure the seriousness of these cases, as they do those of the hemiplegias. As regards the significance of individual symptoms, the post-hemiplegic movements have a bad import; the presence of a microcephalic head or of decided marks of degeneration is unfavorable.

Treatment.—The treatment, so far as the paralysis is concerned, is largely mechanical. The patient is benefited by occasional courses of electrical treatment which stimulate somewhat the nutrition and functions of the muscles. Massage and stretching of the contractured tendons and limbs also are helpful in my experience. The orthopedic surgeon is able to render valuable assistance by occasional overstretching of the contractured limbs and placing them in splints. Tenotomy may also be resorted to with advantage, as I have had occasion to see. The child should be encouraged above all, however, to use the limb as much as possible. He should be taught gymnastic exercises; running, walking, and bicycle-riding are all measures which give great help. When the child's intelligence is good and there is little or no epilepsy, a great deal can be expected in the way of improvement as the child grows older.

So far as the epilepsy is concerned, it should be treated on the same principles as idiopathic epilepsy, except that great care should be had in the use of the bromides; a thorough test must be made in order to determine how much of this drug will sup-

press the fits, and then its use must be graduated in the future in accordance with the knowledge thus obtained. The mental defects of the child can only be helped by proper training of the body and careful education of the mind. The question of operative interference in these cases has of late excited much attention. *A priori* it would not seem as though surgical interference could do good in relieving conditions in which there is destroyed or atrophic tissue. Still the subject must be dealt with empirically, and there have been some results which show that apparently a relief is obtained in a few cases by trephining the skull or by Lannelongue's operation of craniectomy. If there is no microcephalus, if the case is one of hemiplegia with imbecility and epilepsy, the surgeon should simply make an exploratory opening. If he then finds any evidences of compression from the presence of a cyst, this may be very cautiously opened. If there is microcephalus, the linear craniectomy is the operation which is indicated. This has been found to be a very serious surgical measure and should only be resorted to with great caution. In all cases, in operating on children, it has been found that it is imperative that the operation be made as short as possible, and that as little be done at any single operation as is consistent with the indications.

CHAPTER XIX.

DEGENERATIONS AND SCLEROSES.

Degeneration of the brain tissue, as in the case of the cord tissue, leads to sclerosis. Destruction, necrosis, injuries from any cause, lead also to sclerosis. Sclerosis is sometimes a primary change due to a gradual proliferation and increase of connective tissue and neuroglia tissue. We have, therefore, in the brain primary degenerations and scleroses and secondary scleroses. The primary degenerations are bulbar palsy and ophthalmoplegia externa, or, as these diseases are sometimes called, polioencephalitis inferior and polioencephalitis superior; besides these are paralytic dementia, which is a diffuse cortical sclerosis, and disseminated sclerosis; and finally, underlying epilepsy we have, it is believed, a diffuse increase of neuroglia tissue which has been called a neuroglia sclerosis or a gliosis. Secondary scleroses are those which are the result of inflammations, hemorrhages, injuries, embolism, and thrombosis. A secondary sclerosis usually affects certain special areas of the brain and is called a lobar sclerosis. Corresponding to these pathological changes we have certain clinical types of disease. Thus the diffuse cortical scleroses cause what is known as paralytic dementia; polioencephalitis superior and inferior cause bulbar paralysis and ophthalmoplegia; neuroglia sclerosis is perhaps at the basis of epilepsy and other degenerative disorders; lobar sclerosis is the lesion in cases of cerebral palsies of children, such as hemiplegias, diplegias, and paraplegias, and many forms of inco-ordination, motor disturbance, and convulsive manifestations.

Besides the terms that we have used here, there are in medical literature frequently employed the expressions brain atrophy, hypertrophy, porencephaly, chronic cerebritis, tuberous sclerosis, all of which terms are more or less synonymous with what we have already given and are superfluous to our nosology.

There is possibly a primary chronic softening or degeneration of the brain, but if it exists is extremely rare, and occurring in advanced life is probably due to disturbances in the blood supply.

Multiple Sclerosis (Disseminated Sclerosis, Insular Sclerosis, Sclerosis en Plaques Disseminées).

Multiple sclerosis is a chronic degenerative disease characterized by the development of small patches of connective tissue throughout the brain and spinal cord, and by symptoms of paralysis, tremor, disturbances of speech and of the eye muscles, and by a number of other symptoms, very variable in character.

In America the disease is, in the writer's experience, rare. The diagnosis, to be sure, is often made, but only two cases verified by post mortems have been reported by American physicians.

Etiology.—Heredity is a predisposing cause in a small proportion of cases. The disease has been directly inherited; but generally the patient simply inherits a neuropathic constitution. The disease develops in the early part of adult life, oftenest between the ages of thirty-five and forty-five, but it may occur in children, and it is probable that a considerable number of cases developing in adult life are cases in which the seeds of the disease were sown during adolescence. The sexes are about equally affected, the preponderance being rather in favor of the female.

The chief exciting cause of most cases is undoubtedly some infective fever, usually typhoid, diphtheria, small-pox, measles, or erysipelas. It has been known to follow an acute attack of hæmoglobinuria and perhaps of malaria. Myelitis and encephalitis are said to have led to the subsequent development of multiple sclerosis, but this is unlikely. Exposure to cold and wet and traumatisms, especially those which lead to the development of the traumatic neuroses so called, are sometimes the cause of the disease. In fine, acute infections and severe traumatisms involving concussion, co-operating with a neuropathic nervous system, form the most important etiological factors. Syphilis is rarely a cause.

Symptoms.—The disease probably has existed a long time before very notable symptoms develop. Among the first things which the patient observes is a weakness in the limbs, usually in an arm or leg; this is soon accompanied with a tremor which is of a jerky, irregular character. The paralysis slowly extends and the patient becomes partially paraplegic or hemiplegic. He may have some ataxia with the paralysis. At the same time disturbances in vision occur, such as amblyopia, spots before the eyes, nystagmus, and diplopia. Defects in articulation develop; the speech becoming slow and scanning, or "syllabic," as it is called. Attacks of vertigo or apoplectic or epileptic seizures occur. There may be some sensory symptoms, although these are usu-

ally not marked or permanent. When present they consist of pains in the limbs, resembling those of locomotor ataxia, and anæsthesias variously distributed. Mental change is not infrequent and is somewhat characteristic. The patient's mind is rather dull, but his temper is equable and placid. The memory may become weak; very rarely insanity develops.

The disease runs a very variable course, depending upon the seat of the multiple lesions. In typical forms these lesions are distributed throughout the brain and spinal cord, but chiefly in the brain axis, and consequently in these cases the progress of the symptoms is fairly uniform. We have referred to them in the brief outline of the disease; they may be enumerated here again. 1st. Paralysis taking the form of hemiplegia, paraplegia, monoplegia, or some cranial nerve paralysis. 2d. A jerky tremor, often called intention tremor. 3d. Spastic phenomena shown by exaggeration of knee jerks, clonus, and rigidity of limbs. 4th. Cerebral disturbances characterized by vertigo, headache, epileptiform and apoplectiform attacks, and the peculiar mental state referred to. 5th. Nystagmus and disorders of vision and of the optic nerve. 6th. Disorders of articulation or scanning speech. 7th and finally, an irregular and intermittent course to the disease.

We will take up some of these symptoms now in detail. The paralysis is most frequently of the nature of a hemiplegia or paraplegia which is never complete, but rather in the shape of a simple weakness. This paresis is almost always accompanied with a spastic condition, so that there is exaggeration of reflexes and clonus. It is often also accompanied with an inco-ordination, but most frequently of all it is associated with the peculiar "intention tremor" referred to. This tremor is coarse in its character; it ceases when the limbs are at rest and is shown only when voluntary movements are attempted. Thus when the patient tries to raise a glass of water to his lips the jerky tremulous movements increase so that the water is spilled before it reaches his mouth. The tremor affects the muscles of the neck sometimes, so as to cause oscillation of the head; and sometimes the muscles of the trunk, but rarely if ever the facial muscles. The nystagmus, which is another manifestation of the tremor of the disease, is usually lateral and is present in about two-thirds of the cases. Eye palsies involving most often the sixth nerve occur in about one-sixth of the cases. The pupillary and ciliary muscles are rarely affected, and the Argyll-Robertson pupil is not found in the disease. The disturbance of articulation is very common, ranking in frequency with the nystagmus and tremor. In speaking the patient separates the words into distinct syllables, or he may simply separate the words very distinctly from

each other. Sometimes words are dropped or imperfectly pronounced. The trouble seems to be due to inco-ordinate movements of the tongue and other muscles and organs concerned in articulation. The cutaneous disturbances of sensation are not very great. They occur in perhaps one-third of the cases, but are often temporary and are perhaps due to a superimposed hysteria. Severe pain is not a common symptom, but it may exist in the form of neuralgias of the nerves of the limbs or in the shape of girdle pains. The anæsthesias are usually partial and involve only small areas of the limbs. There may be various paræsthesiæ affecting the paralyzed parts of the body. Of the nerves of special sense the eye is most frequently involved, and a study of its condition is most important. The paralyses of its muscles and consequent diplopia have been referred to. Temporary disturbance of vision is not uncommon. Such disturbances consist of constriction of the visual field, amblyopia, and scintillating scotomata. The fundus of the eye shows in some cases a slight degree of neuritis, and in the later stages a true atrophy may be present. One eye is usually more involved than the other, and one eye alone may be affected. The ophthalmoscopic changes in the disc are said to be different from those found in locomotor ataxia and in the neuritis of brain tumors.

Disorders of the visceral centres producing retention of urine, constipation, and trophic disturbances leading to œdema, skin eruption, arthritis, etc., are relatively rare and occur, if at all, in later stages of the disease.

Aborted Types or "Forms frustes" of Multiple Sclerosis.—In some cases the nodules of sclerosis are so limited in number and so peculiarly placed that they give rise to very atypical forms of the disease. Perhaps the most common one is that in which the disease takes the type of a *progressive spastic paraplegia*. The patient suffers from weakness of the lower limbs, accompanied with stiffness, cramps, exaggerated reflexes, and disturbances in the bladder and rectal functions. Anæsthesias, pains, and the girdle symptom may develop. In addition to this, however, a close examination will show some evidence of disease of the optic nerves and perhaps disturbances of the eye muscles. The patient will have nystagmus, diplopia, or other visual disorders. There will also be some attacks of vertigo or of epileptoid convulsions. The combination of the eye symptoms with the progressive paraplegia will often reveal the true character of the disease.

A purely *spinal* form of multiple sclerosis has been described; it is probable, however, that multiple sclerosis never affects the spinal cord alone. A cerebral form of multiple sclerosis has also been described, but this usually runs a course very similar to

that of the true cerebro-spinal malady, and it is unwise to attempt to make any special clinical distinction. Charcot divides the symptoms into cerebral and spinal, and includes under the cerebral the eye symptoms, apoplectic attacks, and speech disturbances; under the spinal symptoms, paralysis and tremor. Tremor, however, is often, if not usually, due to a cerebral lesion.

Pathology.—Grayish nodules are found distributed through the brain and spinal cord. They vary in size from a millimetre to two or three centimetres in diameter (one-twenty-fifth to one

Fig. 184.

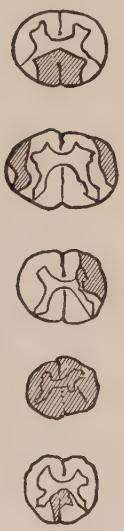


Fig. 185.

Figs. 184 and 185.—Multiple Cerebro-Spinal Sclerosis (Charcot).

inch). They are of firmer consistence than the surrounding brain substance, but are not quite so hard as ordinary connective tissue. They consist microscopically of fibrous tissue which does not seem to be connected with the walls of the blood-vessels as a rule. Very often the axis cylinders of nerves can be seen passing through the lesion. The nodules are found most frequently in the white matter of the brain, more especially in the pons, internal capsule, and centrum ovale (Figs. 184, 185). They rarely begin primarily in the gray matter, but may invade it secondarily. The roots of the peripheral, especially of the cranial, nerves occasionally con-

tain or are surrounded by these sclerotic masses. In the spinal cord they may extend up and down the gray and white matter for a considerable distance, or they may involve the whole cord at a certain level, turning it into a fibrous mass. The blood-vessels surrounding and in connection with these diseased areas show some evidences of thickening and increased vascularity, but no true inflammatory process. The primary pathological change in multiple sclerosis is as yet unknown; many things point to its starting originally from small emboli or thrombi which lead to minute softenings, with a secondary reparative and sclerotic process. The fact that the disease follows infective fevers makes such an origin of it seem probable. On the other hand, pathological anatomy does not yet bear out this view, and it is possible that

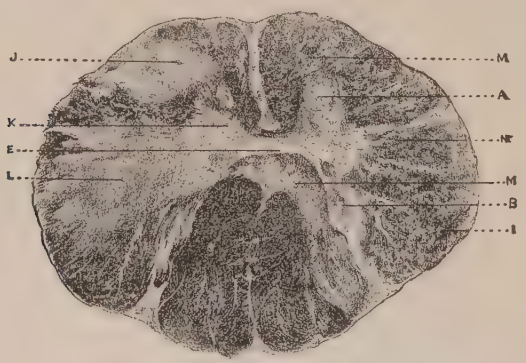


Fig. 186.—Spinal Sclerosis. J, K, L, M, Sclerotic foci; A, anterior, B, posterior horn (Blocq).

the disease begins by a primary degeneration affecting first the myelin sheaths of the nerve fibres, this being followed by a neuroglia and connective-tissue proliferation which ends in the formation of the small islands of sclerosis. An important pathological peculiarity of the process is that while it destroys the myelin sheaths of the nerves, the axis cylinders remain intact for a long time, and consequently conduction of nerve impulses takes place imperfectly, directly through the nodular masses.

Course and Duration.— The disease runs a very irregular course. Its prodromal stage is long and remissions of considerable length occur. The disease may last from five to fifteen years, the average duration being five or six years. Death sometimes occurs from involvement of the nerves of the medulla, but more often from weakness and exhaustion or some intercurrent malady.

Diagnosis.—The diagnosis in typical cases is not very difficult; but as, on the other hand, typical cases are not common, the disease has always to be studied with great care before certainty can be reached. The diagnosis is based upon the slow development of the disease, with attacks of vertigo, weakness, and uncertainty in gait; upon the paralysis of the extremities with intention tremor, ataxia, rigidity, and contractures; upon the disturbances of vision, nystagmus, and the speech troubles. The presence of headache, attacks of vertigo, apoplectiform attacks, and the peculiar mental condition often furnish help. The age of the patient and the cause should also be taken into consideration. The disease must be distinguished from Friedreich's ataxia, spastic spinal paralysis, locomotor ataxia, dementia paralytica, bulbar paralysis, paralysis agitans, chronic meningitis, and hysteria. The points already given and those furnished under the heads of these different diseases must be utilized in making these distinctions. The method of exclusion may be used with advantage in reaching the diagnosis of this protean malady.

Prognosis.—The prognosis, while not favorable as regards the ultimate cure, is somewhat favorable as regards the remission and improvement, and the disease on the whole is not so severe as is locomotor ataxia or the other degenerative disorders.

Treatment.—In the treatment the same measures recommended for other degenerative diseases of the nervous system must be employed. Hygienic measures, electricity, and hydrotherapy have some therapeutic value. Internally the use of large doses of iodide of potassium, the hypodermic injection of arsenic, the administration of nitrate of silver and of quinine and other tonics are advised. A very regular, systematic, and quiet mode of life, combined with the use of iodide of potassium and bichloride of mercury, has produced the best results in my experience, even in cases which gave no history of syphilitic infection.

Hypertrophy and Atrophy of the Brain.

These are both terms which can be dropped from neurological nomenclature as representing distinct disorders. *Hypertrophy* of the brain has been described as a disease occurring in childhood, developing either congenitally or in association with rickets. It has been said to produce much the same symptoms objectively as hydrocephalus. There is not sufficient pathological evidence to justify us in saying that the hypertrophy is really a hypertrophy of the brain, rather than a chronic inflammation or gliomatosis or some other tumor formation.

Atrophy of the brain may be either total or partial. When total, that is, involving the whole of the encephalon, it is congenital and is described under the term microcephaly. A slight

degree of general atrophy occurs during senility and in the later stages of chronic insanity. Partial brain atrophy is either congenital or acquired. When congenital it is due to some intrauterine inflammation, vascular obstruction, or aphasia; and when acquired it is due to some of the numerous causes which have been described under the head of infantile and birth palsies. It will be seen that brain atrophy does not have any place as a distinct disease.

CHAPTER XX.

TUMORS OF THE BRAIN.—SYPHILIS.

The kinds of tumor found in the brain are tubercle, syphiloma, glioma, and sarcoma, which are the common forms; myxoma, carcinoma, fibroma, osteoma, cholesteatoma, lipoma, psammoma, neuroma, vascular tumors including aneurisms, echinococcus, and cysticercus. In fact, all forms of new growths are found in the brain; but the infectious granulomata, tubercle, and gumma and the sarcomatous type of tumors are the most common. As compared with other organic diseases of the central nervous system, brain tumors are rare.

Etiology.—Brain tumors affect males oftener than females, the ratio being about as two to one (644 : 320). Sarcomata alone seem to affect females about as often as males. Brain tumors occur with about equal frequency throughout all ages of life up to about fifty; one-third occur under the age of twenty (Gowers). During childhood tumors are about equally distributed throughout all ages (Starr). One-half of all the tumors of childhood are tubercular; after this come gliomata and sarcomata. The gumma, glioma, and sarcoma begin to be more frequent after the age of twenty. Sarcoma and especially cancer occur in the middle and later ages of life; but brain tumors of any kind are extremely rare after the age of sixty.

To sum up in tabular form, the relative frequency of the different kinds of tumors with regard to age is shown in the following:

Childhood, ...	tubercle, parasites.
Early life, ...	gumma, glioma, parasites.
Early and middle life, . .	sarcoma, glioma, and gumma.
Middle and late life, . .	sarcoma, gumma, cancer.

Heredity has a slight influence in predisposing to brain tumors. Blows on the head and other forms of injury to the cranium are exciting causes in a small proportion of cases.

Symptoms.—The symptoms of brain tumors vary extremely in accordance with the location, the kind of tumor, the rapidity of growth, and the age of the patient. The general course of a

case of brain tumor in an adult is somewhat as follows: The patient first notices a headache which is very intense and persistent and which has exacerbations of frightful severity. With the headache or between the attacks vomiting occurs, which is often not accompanied with any nausea. Sensations of vertigo, annoying paræsthesiæ, and convulsive movements affecting one or more extremities develop, and there may even be general convulsions. The patient finds that his eyesight is weak and progressively deteriorates. The mind becomes more or less disturbed, the mental processes are dull and slow, a feeling of hebetude and incapacity to attempt any mental exertion is present. As the disease progresses the intense pains and vomiting produce weakness and emaciation. Paralyses of various kinds develop. Blindness may ensue. Convulsions of a local or general character become more frequent, and finally the patient becomes bed-ridden and helpless.

The course of the disease is not a steady one, there being often slight remissions, or there may be periods when progress seems to be arrested. After a period of time varying from one to four or five years death occurs from exhaustion or some intercurrent malady.

The symptoms thus very briefly outlined are divided into general and focal. The *general symptoms* are at first those of cerebral irritation and of headache, vertigo, and vomiting, optic neuritis, convulsions, disturbances of speech, and hebetude. Headache occurs in from one-half to two-thirds of the cases; it is very severe and the pains are of a boring or lancinating character; they are so horrible that they often lead the patient to think of suicide. The pains are sometimes periodical, occurring every night or every other day, and suggest by their periodicity a malarial character. They are located sometimes in the brow or in the occiput, while sometimes they are diffused all over the head; they are rather more frequent than otherwise in the neighborhood of the tumor. They are more frequent with cerebellar tumors than with those located anywhere else. They are also frequent with tumors of the mid-brain and of the cerebral hemispheres. They are less frequent when situated in the peduncles and at the base of the brain. The pains are due to the increased intracranial pressure and to irritation of the membranes of the brain by the encroachment upon them of the new growth. Headache occurs in about the same proportion in children and adults, and it does not seem to bear much relation to the kind of tumor, although the pains are generally less with the gliomata, and they are more frequent with rapidly growing tumors whatever their character. With the pains there is often a local tenderness of the scalp and cranium which may be elicited by percussion, and

in most cases there is greater tenderness in that part of the cranium lying over the tumor.

Vomiting is a symptom which is almost as frequent as headache. The vomiting is often of a projectile character and not accompanied with much nausea. Vomiting occurs, as does headache, more frequently with cerebellar tumors. It is associated with rapidly growing tumors such as syphilitic or tubercular neoplasms. Vertigo is a general symptom which occurs in from one-third to one-half of the cases. The vertigo may be slight, such as is often felt from ordinary causes. Occasionally it is very severe and accompanied with forced movements. The severer forms and those associated with forced movements occur with tumors of the cerebellum and the parts closely connected with it. Optic neuritis is one of the most frequent and important of all the general symptoms of brain tumor; it occurs at some period of the disease in at least four-fifths of the cases, more frequently in cerebellar tumors and in those of the midbrain and great basal ganglia. It is rare in tumors of the medulla. It is less frequent and marked in the slow-growing tumors. The neuritis may run a somewhat rapid course and then improve a great deal or even for a time disappear; but ordinarily the course is progressive and it ends eventually in an atrophy of the optic nerve. Hence the examination of the eyes in brain tumors should be made a number of times in order to note the progress of the trouble. Primary atrophy of the optic nerve does not occur in brain tumors. The inflammation almost always affects both nerves, but it may begin with one and subsequently affect the other. The cause of optic neuritis in brain tumors has been the subject of a great deal of discussion and theorizing. There are doubtless different factors entering into the mechanism of its production in different cases, but accepting the modern doctrines of inflammation, we must believe that the most probable and most essential cause is an irritation of the nerve fibres by the products of the growth of the tumor which infiltrate into the sheaths of the optic nerves. There are peculiar mechanical conditions which tend to hold these products within the sheaths and thus enable them to act more energetically as irritants. Simple mechanical processes alone—the tension and pressure—would hardly explain the presence of a genuine inflammation.

Mental defects are almost always present in tumors of the brain. These defects consist in a slowness of the mental processes, a condition of hebetude, a tendency to attacks of somnolence, and sometimes a peculiar childishness and silliness or peculiar mental irritability. The memory is also usually somewhat weakened and the power of attention lessened. Such psychical

defects are more frequent with tumors of the fore-brain and more frequent also with large tumors. General convulsions occur in about one-fourth of the cases and more frequently when the tumors are situated in the cerebral hemispheres and cortex. There may be also apoplectiform attacks, from which the patient recovers in the course of a few days or weeks. More rarely there is a genuine apoplexy from the bursting of a blood-vessel in the neighborhood of the tumor.

The speech disturbances are most marked in tumors which affect the pons and medulla and the origin of the cranial nerves. Such speech disturbances, when characteristic, are shown by a confluent articulation, that is to say, the patient runs the syllables together.

The cranial temperature in brain tumors is in most cases somewhat raised as compared with the normal (Gray, Mills, and Lloyd). The elevation may be several degrees above the normal. The normal average scalp temperatures (Gray) are from 92° to 94.5° F., being somewhat higher over the frontal and parietal than over the occipital regions. In brain tumors the temperature has been found raised to 95°, 96°, and 98°. The value of thermometric observations, however, in the symptomatology of brain tumors is somewhat doubtful, owing to the variability in the normal temperature and the difficulty of getting accurate records.

Focal Symptoms.—The symptoms of brain tumor are produced in part by the irritation and pressure, in part by destruction of the normal tissues. The symptoms are also modified by variations in the vascularity of the tumors, and perhaps to some extent by direct irritation from products secreted by the tumor cells. Having by a study of general symptoms arrived at a fairly certain diagnosis as to the presence of a tumor, it is necessary to corroborate the diagnosis and to localize the lesion by an examination of the symptoms which are the result of irritation or destruction of certain particular parts of the brain; these are called the focal symptoms. For purposes of special or local diagnosis we divide the brain into the following parts or areas (Knapp): 1. The præfrontal, which includes all that part lying in front of a line that extends from the upper end of the ascending branch of the fissure of Sylvius directly up at right angles to a horizontal line between the frontal and occipital poles of the brain (see Fig. 187). This region includes probably centres for the movement of the head and eyes, but it is chiefly concerned with the higher intellectual processes; its under surface lies on the orbital plate of the frontal bone and upon the right olfactory lobes. 2. The central region, which is bounded in front by the vertical line just described, behind by a line passing down from the anterior end of the parietal fissure to the fissure of Sylvius, and

above by a line that bounds posteriorly the post-central convolution. 3. The parietal lobe. 4. The occipital lobe. 5. The temporal or temporo-sphenoidal area. 6. The corpus callosum. 7. The great basal ganglia and capsules. 8. The corpora quadrigemina, deep marrow, and pineal gland. 9. The crura cerebri. 10. The pons and medulla. 11. The cerebellum. 12. The basal

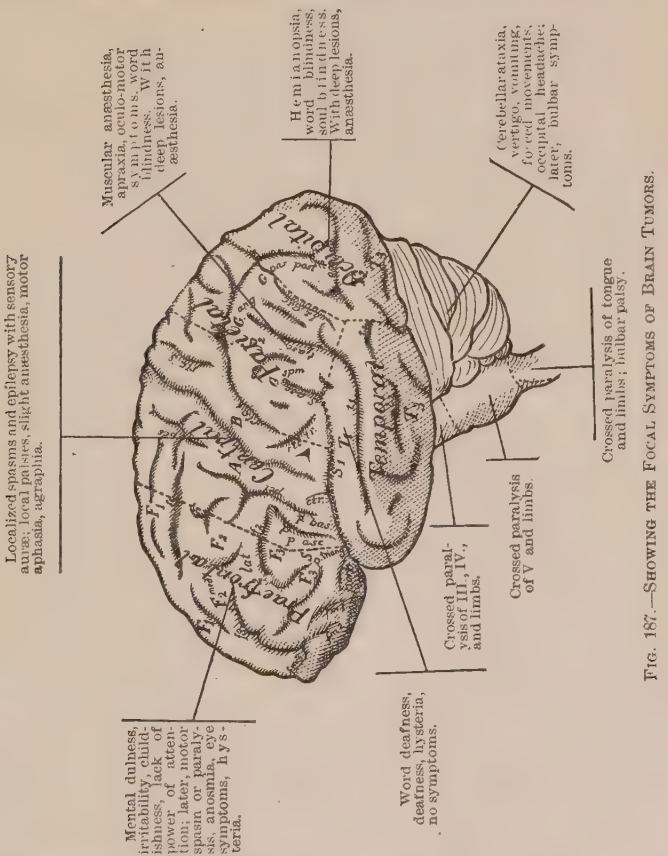

Localized spasms and epilepsy with sensory aura; local palsies, slight anæsthesia, motor aphasia, agraphia.
Muscular anæsthesia, apraxia, oculo-motor symptoms, word blindness. With deep lesions, anæsthesia.
Hemianopsia, word blindness, soul blindness. With deep lesions, anæsthesia.
Cerebellar ataxia, vertigo, vomiting, forced movements, occipital headache; later, bulbar symptoms.
Crossed paralysis of tongue and limbs; bulbar palsy.
Crossed paralysis of V and limbs.
Crossed paralysis of III, IV, and limbs.
Word deafness, deafness, hysteria, no symptoms.
Mental dulness, irritability, childishness, lack of power of attention; later, motor spasm or paralysis, anosmia, eye symptoms, hysteria.

FIG. 187.—SHOWING THE FOCAL SYMPTOMS OF BRAIN TUMORS.

surface of the brain. The boundaries of most of these areas are indicated better by the figure than by a description. They correspond to some extent with the cerebral lobes, but not entirely so, since the frontal and parietal lobe divide between them the central area.

1. Tumors of the præfrontal area. Tumors in this area often show no particular localizing symptoms, and this part of the brain

is consequently put down as a latent one; nevertheless, in a good proportion of cases tumors here produce peculiar mental disturbances that, taken in conjunction with the general symptoms, enable us to make a local diagnosis. The symptoms are peculiar mental hebetude, childishness, irritability, often a kind of silliness and emotional weakness, a tendency to laugh and cry and to get angry at trifling causes. The entire character and temperament of the man are sometimes changed. Besides this, owing to implication of the olfactory nerve, there may be loss of the sense of smell on one or both sides; implication of the optic nerves will cause hemianopsia and optic neuritis. If the tumor involves the orbit there will be paralyses of the ocular muscles and protrusion of the globe of the eye. If the tumor grows backward there is gradual invasion of motor centres with irritation, showing itself by spasms, convulsions, and later by paralyses.

2. Tumors of the central region. It is in this area that we are often able to make the closest and most accurate diagnosis of the localization of new growths, owing to their involvement of the different motor centres. Through this involvement these centres are at first irritated, with the result of producing local spasms or Jacksonian epilepsy. Such spasms are often preceded by sensory symptoms or auræ. As the tumor grows the area of involvement becomes larger, spasms become more diffused, and general convulsions may finally appear, with hemiplegia. The motor disturbances are not infrequently accompanied with sensory disorders. These may be simply feelings of numbness or prickling which either are permanent or simply precede spasms, or there may be hemianæsthesia of a moderate degree to pain, touch, and temperature. The muscular sense also may be somewhat involved. In case of slight sensory involvement the capacity for localizing sensations seems to be most implicated. Besides the symptoms mentioned, there may also be motor aphasia and agraphia. The exact localization must be worked out with the help of the figures and descriptions given under anatomy.

3. Tumors of the parietal area. The symptoms produced by tumors in this area may be very slight. The most characteristic are disturbances of muscular sense which occur when the supramarginal gyrus is affected, and word blindness which occurs when the angular gyrus and inferior lobule are affected. When the tumor is higher up near the longitudinal fissure, the muscles of the lower limbs may be involved, and if the tumor encroaches upon the central area spasms and paralyses of various muscular groups ensue. The cortical representation of the third nerve is thought to be in the neighborhood of the angular gyrus, and some cases have been reported in which paralysis of this nerve resulted from tumors in that area.

4. Occipital lobes. Tumors in this region, if situated in the cuneus and first occipital convolution, produce homonymous hemianopsia. If the tumor involves the other parts of the occipital lobe and the cuneus is not seriously involved, there may be a condition known as soul blindness or incapacity to understand the nature of the things which one sees. If the tumor extends up chiefly toward the angular gyrus, there may be word blindness, along with some hemianopsia. If the tumor extends farther forward into the parietal lobe, there may be hemianæsthesia, hemiataxia, and perhaps a little hemiplegia owing to involvement of the fibres of the internal capsule.

5. Temporal area. The temporal or temporo-sphenoidal area on the right side is very nearly a latent one. On the left side tumors involving the posterior part of the first and upper posterior part of the second temporal convolution produce word deafness. Tumors in either lobe when large and extending well down toward the base may produce attacks of vertigo or forced movements, owing probably to irritation of the internal ear. Tumors that involve the hippocampal convolution and the uncus may produce perhaps some disturbances in the senses of smell and taste.

6. Tumors of the corpus callosum. Tumors situated in this area are very rare. Their symptoms have been thought to be somewhat characteristic; but in the writer's experience they correspond closely with tumors situated in the third ventricle and lateral ventricles of the brain; in other words, tumors which, beginning in the central parts of the brain, gradually extend outward toward the periphery. The symptoms credited to tumors of the corpus callosum are, first, the general symptoms of brain tumor, to which there are superadded a gradually developing hemiplegia with later a paraplegia. At the same time there is a great deal of mental dulness, stupidity, and drowsiness; the patient often sits for hours mute, refusing to speak, or lies in a half-somnolent condition. There are no paralyses of the oculomotor nerves or of the other cranial nerves. There is no anæsthesia. The disease gradually progresses and the patient dies in coma.

7. Tumors of the great basal ganglia and the capsule (the optico-striate region). The general symptoms of tumors of this region resemble in many respects those of tumors of the corpus callosum. The stupidity, however, may be less marked. There is usually a progressive hemiplegia which may be accompanied with anæsthesia and sometimes with choreic movements, if the tumor involves the optic thalamus and adjacent part of the capsule. Tumors of the caudate nucleus alone and of the lenticular nucleus alone seem to give rise to no special symptoms, and these

regions are regarded as latent. Tumors of the anterior three-fourths of the optic thalamus alone may cause no special symptoms, but in some cases there occur peculiar choreic or athetoid movements. These, however, are probably due to irritation of the fibres of the internal capsule. If the tumor involves the posterior part of the optic thalamus and adjacent areas, there will be a hemianopsia, which may be distinguished from the hemianopsia due to lesions in the occipital lobe by the presence of the *hemiopic pupillary reaction*, that is to say, a ray of light thrown in upon the insensitive part of the retina will not produce a reflex contraction of the pupil.

8. Tumors of the corpora quadrigemina, deep marrow, and pineal gland. The characteristic symptoms, as shown by Nothnagel, of tumors of this region are inco-ordination, forced movements, and oculo-motor palsies. Together with these there may be hemianopsia or blindness due to destruction of the primary optic centres. It is possible that some degree of deafness or hemi-deafness may be produced by the involvement of the posterior tubercles of the corpora quadrigemina.

9. Tumors of the crus. Tumors of the crura cerebri are extremely rare. When present, they cause hemiplegia and perhaps a hemianæsthesia, with paralysis of the third nerve upon the same side as the lesion; in other words, a crossed paralysis.

10. Tumors of the pons and medulla. Tumors in this area necessarily produce very varying symptoms in accordance with their size and location. If the tumor is in the pons it will cause, if situated high up, a palsy of the third nerve upon one side and hemiplegia on the opposite side. If lower down there may be a palsy of the fifth nerve on one side and hemiplegia on the other side. If the tumor is extensive it may produce not only a hemiplegia, but a hemianæsthesia. If situated somewhat superficially and on the lateral edge of the pons involving the peduncles, there will be forced movements of the body, either toward or from the seat of the lesion. If the tumor is in the medulla it will produce hemiplegia and hemianæsthesia, with paralysis of the hypoglossal nerve or perhaps some other cranial nerves upon the same side. If large and involving both sides of the medulla, there may be the general symptoms of a progressive bulbar paralysis. One peculiarity of tumors situated in the pons is that they sometimes produce a conjugate deviation of the eyes which is away from the side of the lesion. In this respect the symptoms differ from conjugate deviation produced by lesion in the cerebral hemispheres where the head and eyes are turned toward the side of the lesion.

11. The general symptoms of tumors of the cerebellum are, as we have already said, more pronounced than those of tumors of

other regions; we more frequently have headache, vomiting, vertigo, and optic neuritis from neoplasms here. If the tumor is situated in the lateral lobes of the cerebellum, no localizing symptoms develop until the tumor becomes very large so that it presses upon the medulla or other adjacent regions. When the tumor is in the middle lobe a peculiar ataxia develops, known as cerebellar ataxia. The gait of the patient is a reeling one like that of a drunken man, or in walking he takes short steps and spreads his legs as if in fear of falling. This has been called the titubating gait. Besides this, severe forced movements may occur which usually throw him sideways or perhaps forward, very rarely backward. Secondary symptoms from pressure on the medulla often develop in tumors of the middle lobe; such symptoms being glycosuria and disturbance of the functions of the cranial nerves. Late in the disease hemiplegia and paraplegia and bulbar symptoms may develop from extreme pressure. There may be also hydrocephalus due to pressure on the veins of Galen and obstruction of the return flow of blood from the central arteries of the brain.

12. Tumors of the base of the brain. Tumors situated in the anterior fossa produce symptoms very much like those described under the head of tumors of the præfrontal area, but there is necessarily destruction of the olfactory lobe and there is more apt to be involvement of the optic and oculo-motor nerves and of the tissues of the orbit.

Tumors of the middle fossa. Tumors sometimes involve the hypophysis. Such condition has been found in cases of acromegaly, and it has been suggested that the tumor of the hypophysis is the cause of the disease referred to; but such cannot be the case, since a number of tumors of this region have been described in which none of the symptoms of acromegaly were present. Tumors of this region and of the interpeduncular space produce symptoms such as would naturally result from pressure on the optic chiasm, and it is mainly the early presence of optic neuritis and of peculiar forms of hemianopsia which differentiates lesions in this area from those in the anterior fossa.

Multiple tumors. About one-seventh of all brain tumors are multiple. Hence in making a diagnosis of the localization of tumors this fact must be borne in mind. The tumors which are most frequently multiple are tubercle, cancer, and melanotic growths.

Pathology.—Tubercle is a form of tumor found oftenest in children and is altogether the most frequent of brain tumors. It is more often located in the cerebellum, but may appear in the pons or other parts of the brain. It may be a single or, as it is then called, a solitary tubercle, or there may be a multiple

growth. The tumor is irregularly round in shape and varies in diameter from one and a half to two inches. It has a grayish-yellow appearance externally; internally, a yellowish or cheesy look. It is not vascular, but is often surrounded by softened or inflamed tissue. There may be an associated meningitis. The tumors, when solitary, usually start from the central parts of the brain, but they also develop on the meninges of the convexity, particularly in the parietal region, and sometimes they develop also at the base. Tubercle always arises from infection by the tubercle bacilli which are carried by the blood to the brain. The tumors develop usually from some infectious focus, starting in a blood-vessel of the pia mater. Microscopically the tumor shows the ordinary appearances of tuberculous growths. It contains in its periphery many round cells, nuclei, and giant cells. In the centre there is usually an amorphous substance, the product of degeneration and the breaking down of the ordinary substance of the tumor. The characteristics of the growth are the presence of the round cells and giant cells, the caseation and softening of the centre, and the absence of vascularization, with the presence of the bacilli.

Syphiloma or gumma. Gummatous tumors of the brain are usually associated with syphilitic meningitis or some other form of cerebral syphilis such as endarteritis and perhaps inflammation of the cranial nerve roots. Syphilitic growths are usually found upon the brain surface, oftenest on the base, next upon the convexity of the frontal and central convolutions. The process appears either in the form of a somewhat distinct tumor or in the form of an irregular thickened exudate lying upon the surface of the brain and forming what is called gummy meningitis. The gummata may attain great size. They start usually from the pia mater and are due, as in the case of tubercle, to the irritative action of some infective organism. The gumma is irregular in shape; it has a somewhat thick grayish periphery and often a yellowish centre, the appearances differing with the age of the tumor. Microscopically it is found to consist of small round cells and spindle cells with various broken-down nerve-tissue elements. It presents in the interior the evidence of cheesy degeneration, somewhat like that in tubercle, but less marked. There is a peculiar development of fibrous tissue in the syphilitic growths which distinguishes them somewhat. Besides this, the blood-vessels are numerous in the periphery and show evidences of endarteritis and periarteritis. The distinctions between gumma and tubercle are the less amount of cheesy degeneration in the centre of the former, its more irregular appearance, the presence of arteritis and vascularization, the absence of giant cells and of tubercle bacilli.

Actinomycosis is a form of infectious tumor which sometimes extends from the face and neck into the brain, leading to inflammatory processes, however, rather than true tumors. No other neoplasms of infectious origin attack the brain unless glioma be found to be of that nature.

Glioma may occur in any part of the brain, but is most frequently found in the cerebrum. It is the only tumor which is peculiar to the nervous centre, being developed from the neuroglia tissue which forms the supporting structure of these centres. Glioma originates in the white matter of the nerve centre and not from the membranes or fibrous structures. It may grow to a very large size and is the form of brain tumor which becomes the largest. Gliomatous tumors measure from three to eight or more centimetres in diameter. In appearance the glioma can be scarcely distinguished from the brain substance itself, but usually looks like either pale or congested gray matter, or it may have a yellowish or gelatinous appearance. The tumor is very vascular and it may show the results of hemorrhages. The central part sometimes breaks down, forming cavities or cysts. The tumor may grow very rapidly, infiltrating the normal tissue. In those cases there is hardly any definite boundary between the tumor and the normal tissue. In other cases the tumor grows slowly, but rarely if ever becomes encapsuled. Microscopically it is found to consist of small cells with delicate fibrous prolongations, these being the glia cells. The tumor is very vascular and its whole appearance is suggestive of an inflammatory process rather than a new growth; the inflammatory process being one in which the neuroglia tissue reacts to the inflammatory irritant. Gliomata may undergo certain changes, *e.g.*, a mucous degeneration of the cells takes place, forming a myxo-glioma. When there is with the neuroglia-cell proliferation a rich proliferation of round cells from the connective tissue it is called a glio-sarcoma. When the tumor is situated near the surface, involves the membranes, and grows slowly, with an increase in fibrous tissue, it is called a fibro-glioma. When the gliomatous growth is very firm and hard, the fibrous portion of the glia tissue predominates; it constitutes a nodule such as is found in multiple sclerosis, and these hard gliomata are sometimes called neuro-gliomata.

Sarcoma. The sarcoma and its various modifications form perhaps the most important and almost the most frequent of the brain tumors. The sarcoma is a tumor of connective-tissue origin; it develops, therefore, from the brain membranes or from the sheaths of the blood-vessels. Sarcomas may be single or multiple. They may be of all shapes and they grow to very varying sizes. They often develop a capsule. They may be either primary or secondary. Their growth is often rapid. They are

white or grayish in appearance or may be somewhat yellowish, dependent on the predominance of the different kinds of cells and blood-vessels. Microscopically they are made up of small round cells, spindle cells, and other cells of various sizes and forms. They contain often considerable fibrous tissue. They contain blood-vessels, but are not richly vascular. The essential characteristic of the sarcoma is the rich development of round cells and spindle cells; in other words, its rich cellular contents. Sarcomata are peculiar in undergoing many modifications: thus sometimes fibrous tissue develops largely and the tumor is called a fibro-sarcoma; sometimes the tumor undergoes mucous degeneration and is called a myxo-sarcoma. There may be a breaking down of the centre with the formation of cysts. There may be a development of pigment. Not infrequently a sarcomatous process invades a glioma and we have a mixture of a sarcoma and glioma. Sarcomatous tumors sometimes have an alveolar structure. These tumors contain endothelial cells derived from the lymphatics and are called endothelioma. When sarcomata develop from the dura mater and are slow in growth there may be calcareous deposits in them and they are called psammomata.

The fibroma is a very rare brain tumor, unless the pachionian bodies, when enlarged and hardened, may be so considered.

Osteoma is not particularly rare, developing in the form of bony plates in the dura, falx, or tentorium. Osteomata in the brain substance are merely pathological curiosities.

Enchondromata, lipomata, and angiomata are rare and have no practical importance.

Occasionally neuromata or false neuromata are found developing on the roots of the cranial nerves.

Cancer is relatively a very rare affection of the brain, especially as a primary development. It usually arises from the membranes of the brain. Cancer is not infrequently multiple and is usually of the soft or colloid character.

Parasitic growths. Parasitic tumors are extremely rare in this country. The only forms which are found are the echinococcus and the cysticercus cellulosæ. The echinococcus produces hydatid cysts which may be large or small, few or many, and are usually all upon the surface of the brain. They are much rarer than the cysticerci. These form cysts which are usually multiple, slow in growth, lie upon the surface of the brain or in the ventricles, are encapsuled, and show no symptoms (Fig. 188).

Aneurisms are anatomically tumors; but clinically they present some special symptoms and hence are described separately.

Diagnosis.—It is necessary first to make the diagnosis of the presence of the tumor, next of its location, and finally of its nature. The existence of a brain tumor is determined by the

presence of the characteristic general symptoms—headache, vomiting, vertigo, optic neuritis, mental disturbances, and progressive course. The physician must bear in mind the possibilities of meningitis, abscess, lead-poisoning, hysteria, and paretic dementia. Very often a localized basilar meningitis of syphilitic or tubercular origin simulates closely the presence of a tumor. Besides the general points referred to, in estimating the probabilities of the existence of a tumor we must bear in mind the age of the patient and the existence of a tuberculous or syphilitic history, the history of an injury, of local tenderness, and of rise of cranial surface temperature, and the presence of some new growth

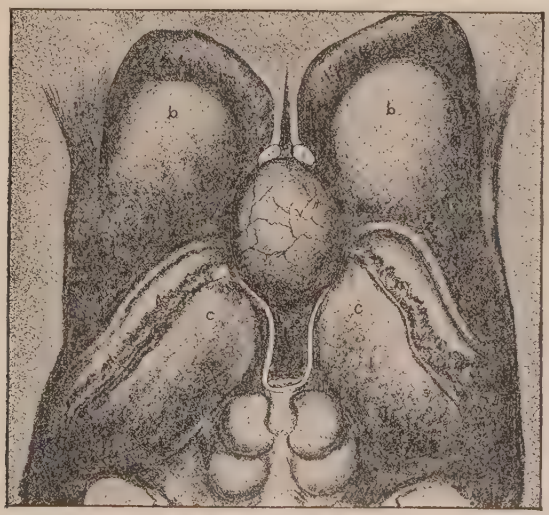


FIG. 188.—ECHINOCOCCUS CYST OF THE THIRD VENTRICLE.

in other parts of the body, particularly about the neck or thorax or in the lungs.

The diagnosis of the location of the tumor is based upon the rules already given in regard to local diagnosis. The diagnosis of the nature of the tumor can often be made and should be attempted. In children, for example, the chances of the tumor being tubercular are very great, particularly if there is a scrofulous diathesis or tubercular disease elsewhere. Syphilitic tumors of the brain are almost always accompanied with or preceded by manifestations of external syphilis. Gliomatous tumors occur in childhood and early life. They produce, as a rule, fewer irritative phenomena and are accompanied by remissions and by apo-

plectic or pseudo-apoplectic attacks due to the vascular nature of the tumor. Carcinomata occur late in life and are usually secondary.

Prognosis.—In extremely rare cases tumor of the brain appears to stop growing and become encapsulated and atrophied. Such tumors are of a tubercular or syphilitic, perhaps sometimes of a sarcomatous character. As a rule, the brain tumor grows steadily and the symptoms of the disease become more pronounced until death occurs. The prognosis is best for tubercle in children and gumma in adults. It is worse in those cases of glioma and sarcoma which have a rapid course. In fact, the sooner serious symptoms develop, the more rapid the general development of the disorder, the sooner does a fatal termination come. The disease lasts on an average two or three years, ranging from a month to eighteen years.

Treatment.—Something can be done in cases of tuberculous tumors, syphilitic tumors, and possibly in the sarcomatous variety. In tuberculous tumors a general constitutional and strengthening treatment must be resorted to; fresh air, tonics, and a large amount of food being the main reliance. The utility of any form of tuberculin is as yet doubtful. In syphilitic tumors much can be done by the usual vigorous antisyphilitic treatment. In sarcomatous tumors, if they are suspected, some help may be obtained from the internal use of arsenic. Symptomatically we must give such drugs as antipyrin, phenacetin, antifebrin, codeine, and perhaps morphine for the relief of pain. The ice-cap and leeching often help the headache also. Should convulsions develop the bromides should be used, just as in idiopathic epilepsy.

In cases in which the location of the tumor can be made out, the question of surgical interference should be considered. The percentage of cases in which surgery can help is extremely small. It will only include those cases in which the tumor can be located; of those which can be located, only those which are in an accessible region, and finally, of those which are in an accessible region, it includes those which are either superficial, or, if lying in the brain substance, are more or less encapsulated. The removable tumors of the brain amount to less than five per cent. They are, in particular, the sarcomatous, syphilitic, and tubercular tumors lying in the central or occipital areas. In many cases where there is some doubt as to the localization an exploratory trephining is justifiable, and in a few cases in which it is known that the tumor cannot be removed the trephining for the simple purpose of relieving pressure is justifiable. It is probable that in adults some tumors from the anterior and middle fossa of the brain can be removed. In children tumors cannot be removed successfully

from the cerebellum; in adults it is perhaps possible. Operations for tumors should be undertaken as early as possible; this is a fact on which too much stress cannot be laid.

Intracranial Aneurisms.

Intracranial aneurisms are of two kinds—"miliary" and those of large size. The miliary aneurisms are minute dilatations of the vessels and are always multiple; they have been described under the head of cerebral hemorrhage. Large aneurisms affect only the large cerebral arteries at the base of the brain. The arteries are affected in the following order: middle cerebral, basilar, internal carotid, and anterior cerebral. The anterior and posterior communicating and vertebral arteries are occasionally involved, the posterior cerebral and inferior cerebellar very rarely (Gowers).

Etiology.—Males are affected slightly oftener than females. Aneurisms occur at all ages from ten to sixty; before ten and after sixty they are extremely rare. Heredity occasionally plays a part in predisposing to cerebral aneurisms. The exciting causes are embolism, especially when the emboli contain microbes; syphilitic disease, injuries, and in rare cases senile degeneration.

The *symptoms* are very indefinite; they resemble to a considerable extent those of tumor at the base of the brain; headache and vertigo, mental dulness and irritation, cranial-nerve palsies, and occasionally hemiplegia and convulsions are noted. Optic neuritis is rather rare. In a few cases the patient is conscious of a murmur or recognizes the pulsating sensation in the head. Sometimes when the aneurism is in the vertebral artery a murmur can be heard between the mastoid process and the spinal column (Moser).

The *diagnosis* is often difficult; it is based on symptoms of tumor at the base of the brain pressing on cranial nerves and on motor or sensory tracts. The effect of carotid compression should be tried.

The *prognosis* is not good. In perhaps the majority of cases a rupture of the vessel occurs in a few years; however, rupture is not the inevitable event, and sometimes the disease becomes stationary or undergoes spontaneous cure.

The *treatment* of the disease, if it can be recognized, is the same as that for aneurism elsewhere so far as drugs are concerned; surgically the common carotid may be tied and perhaps the vertebral if the aneurism is believed to be connected with that artery or with the basilar.

Syphilis of the Nervous System.

Syphilis is an extremely important factor in the causation of the organic diseases of the nervous system. Nervous syphilis makes up over ten per cent of all the hereditary forms, and while in adult life the specific virus attacks nerve centres relatively less often, yet it is a factor whose importance is very great. In the previous descriptions of nervous diseases we have referred to the syphilitic element in connection with etiology and pathology; but syphilis produces upon the nervous system certain forms of disease which are characteristic, hence it is best to take a brief survey of the effects of this infection independently. Syphilis is beyond much question the result of an infection by a microbe, and the result of the activity of this microbe upon the nervous centres is to produce a condition which is really a form of inflammation. The reader can best understand syphilitic disorders, therefore, by remembering that they are all forms of an inflammation. The syphilitic infiltration is simply an exudative inflammation with a specific exudate; the syphilitic gumma is a deposit of this exudation analogous somewhat to an abscess. Syphilis attacks chiefly the membranes of the brain and spinal cord and the blood-vessels of these organs; in particular the base of the brain and the blood-vessels that supply this region are affected. Syphilis acts on the nervous centres in four ways: it produces meningeal exudation and inflammation, it forms gummatous tumors, it causes arteritis, and it leads to degenerations.

Etiology.—A neuropathic constitution probably predisposes to the development of nervous syphilis. The age at which it occurs most frequently is between twenty and forty, but it may occur at all periods of life from infancy up. It attacks men oftener than women in the proportion of about seven to one. Hereditary syphilis makes up about three per cent of the cases. Inadequate and improper treatment of the disease at first favors the development of nervous syphilis later. The neglect in particular of the early use of iodide of potassium tends to render a person more liable to have the nervous centres attacked. The use of alcohol, excesses in the way of severe bodily exercise, severe mental strain, and overwork predispose to the development of the disease; injuries undoubtedly have a similar effect. The time after the infection when the disease is most apt to occur is the third year, but it is not infrequent between the second and the tenth years, and it is possible for nerve syphilis to develop from within a few months up to thirty years after the infection.

Symptoms.—Since syphilitic changes may attack any part of the nervous centres, the symptomatology of nerve syphilis is

necessarily a varied one. The reader can perhaps best understand the way in which the disease acts by having presented first a series of tables showing on the one side the clinical symptoms, on the other the chief anatomical changes that underlie them. The first table presents the symptoms of syphilis of the brain, which is unquestionably the most common form. The next table shows the symptoms of syphilis of the cerebro-spinal system, a form which ranks second in frequency. Third we have syphilis of the spinal cord alone, which is somewhat rarer, and last and rarest of all we have syphilis of the nerves. In addition to these four forms of syphilitic manifestation we have two diseases which are acknowledged to be sequelæ of syphilis and which are called post-syphilitic degenerative processes.

I. Syphilis of the Brain.

Clinical Symptoms.	*Anatomical Change.*
Severe headache, vomiting, vertigo, mental dulness, and irritability, attacks of somnolence or coma, convulsions, cranial-nerve palsies, optic neuritis, hemiplegia, polyuria, and polydipsia.	Gummatous inflammation of the base involving nerve roots, or gummatous inflammation of convexity, arteritis, and phlebitis.

II. Cerebro-spinal Syphilis.

Many of the brain symptoms as above, spastic paraplegia with spinal pains and involvement of sphincters.	Gummatous basilar meningitis ; diffuse, disseminated, or localized meningo-myelitis.

III. Spinal Syphilis.

Paraplegia with pains, Brown-Séquard paralysis.	Meningo-myelitis, gumma, localized softenings from obliterative arteritis.

IV. Syphilis of Nerves.

Cranial-nerve palsies, cauda equina symptoms, local palsies of peripheral nerves.	Root neuritis, gummatous neuritis.

V.—Post-syphilitic Degenerative Processes.

Locomotor ataxia; general paresis.

Taking up these different forms of nerve syphilis in order, I will give some further details with regard to each of them.

I. Syphilis of the brain in its most common form shows itself by a gradual development of severe and persistent headache. This is usually associated with vertigo, sometimes with nausea and vomiting. After the headache has developed and has lasted for a time, or even without much delay, there comes on sometimes an attack of hemiplegia. Preceding the hemiplegia, or in some cases without the hemiplegia, there are paralyses of the cranial nerves, more especially of the nerves of the eye. Optic neuritis is somewhat frequent. There may be, before any paralyses develop, attacks of epileptic convulsions, either general or partial.

Without any paralyses or with simply cranial-nerve paralyses there may develop attacks of somnolence and coma. Even if such attacks do not appear the patient often shows a mental irritability and weakness, a slowness of the reasoning process, and incapacity to fix the attention such as is observed in connection with brain tumors, only with nerve syphilis these symptoms are not usually so marked. Polyuria and polydipsia are symptoms which are occasionally met with. It will be seen that the syphilitic poison produces very various manifestations when it attacks the brain. The characteristic features are this variability in the symptoms and their remittent character. Elaborate systems of clinical classification might be made out of these various groups, but it will be sufficient for the present purpose to call attention to the fact that the intense headaches, optic neuritis, cranial-nerve palsies, attacks of somnolence and coma, and hemiplegia associated with some of the foregoing symptoms are characteristic of most of the forms. The reason for the peculiar symptoms in brain syphilis is manifest when it is known that the lesion most commonly found underlying them is a gummatous meningitis which has a special predilection for the base of the brain. In particular it seems to attack the interpeduncular space and the neighborhood of the optic chiasm and the surface of the pons Varolii. This gummatous meningitis consists of a syphilitic inflammatory exudate which surrounds, presses upon, and injures cranial nerves, attacks the arteries of the base, producing an obliterating arteritis and consequent softenings, with the hemiplegia which is so often a manifestation of the disease. Much less frequently the inflammatory process attacks the convexity, and then it assumes the form of a gummatous patch which produces cortical irritation with headaches, mental disturbances, and convulsions.

II. The next form of nervous syphilis is the cerebro-spinal. In this we have almost exactly the same conditions and symptoms so far as the brain is concerned; but in addition there are symptoms due to more or less diffuse syphilitic inflammation of the pia mater of the spinal cord. The syphilitic process often extends into the spinal cord, producing an obliteration of the arteries and softening with the symptoms of a transverse or a central myelitis. Thus we have combined the symptoms of cerebral syphilis and paraplegia with, as a rule, considerable pain in the back, produced by the involvement of the meninges.

III. The third type of syphilis is the spinal form. The symptoms in spinal syphilis are usually those of a transverse myelitis, involving, in the writer's experience, most often the lower part of the dorsal and upper part of the lumbar cord. This myelitis usually comes on rather slowly with the ordinary symptoms of a

chronic or subacute transverse myelitis. It is probable that syphilis is a much more frequent factor in the production of so-called transverse myelitis than is usually supposed. The anatomical process underlying it is that of a meningitis which passes along the septa into the substance of the cord, involves the arteries of the cord, and produces a more or less complete softening of the part. The only truly inflammatory process, therefore, is that which is produced in the meninges, connective tissues, and arteries. The anatomical changes in the cord substance are mainly those of softening with reactive inflammation. Spinal syphilis may show itself also by the development of gummatous nodules which grow from the meninges, press upon the cord, and produce the symptoms of a spinal tumor. Spinal syphilis may also develop itself in three or four different foci, producing the symptomatology of disseminated myelitis.

IV. Syphilis of the nerves. Syphilis rarely affects the peripheral nerves; there are, however, occasional deposits of syphilitic exudate producing the ordinary symptoms of irritation and compression of nerves. There is said to be a form of multiple neuritis produced by syphilis, but its actual existence has not yet been absolutely demonstrated. Syphilis has been known to attack the roots of the cranial nerves, producing a root neuritis; and it is very apt to attack the roots of the spinal nerves when the spinal membranes are involved.

V. The post-syphilitic degenerative processes are locomotor ataxia and general paresis. Occasionally it happens that the syphilitic deposits in the spinal cord may produce lesions somewhat like those of locomotor ataxia, and in this case there will be a train of symptoms which also resemble this disease. In true locomotor ataxia, however, the process is never in any sense a syphilitic one. The syphilitic poison seems to so affect the nervous centres as to predispose them to the peculiar degeneration characteristic of tabes. Syphilis may also produce a chronic meningo-encephalitis which will manifest itself by symptoms resembling to a considerable extent general paralysis; but it is very generally conceded that true general paresis is not a syphilitic disorder. Syphilis, however, seems to predispose to it, just as it does to locomotor ataxia.

Hereditary Syphilis.—Inherited syphilis will lead to anatomical changes and clinical manifestations resembling in all respects those of acquired syphilis. Inherited syphilis, in other words, may produce headaches, cranial-nerve palsies, hemiplegia, epilepsy, mental disorders, and paraplegia. The disease probably is the cause of a considerable proportion of the cases of chronic hydrocephalus and of many of the cases of so-called tubercular meningitis. The peculiarities of hereditary syphilis show them-

selves rather more in diffuse symptoms such as would be attributed to a meningitis of the convexity; in other words, convulsions and mental weakness are rather more frequent, while hemiplegia and cranial-nerve palsies are comparatively rare. Hereditary syphilis also very rarely indeed attacks the spinal cord, although it is not unlikely that it is a factor in the production of some of the hereditary scleroses of that organ. Hereditary syphilis develops at any time from birth to the eighteenth year, but most commonly under the age of five years.

Pathology.—I have already given some indications of the pathological changes produced by syphilis. The disease affects the nervous system (1) by producing a meningitis with infiltration, (2) by producing gummatous masses, (3) by producing an inflammation of the arteries, and (4) by so influencing the nervous system as to lead to the development of degenerative diseases. Of all these forms of anatomical change it is the arteries that are most often affected, and particularly the arteries at the base of the brain.

Syphilitic meningitis is characterized by the proliferation of round cells and the preponderance of an exudate which has a tendency to infiltrate into the nervous tissues. The anatomical characteristics of the syphilitic gumma must be studied in special text-books. The inflammation of the arteries attacks first the external coat and adventitia, producing there an enormous multiplication of round cells. The external coat becomes weakened, and as a result there develops beneath it, between the intima and the elastic layer, another exudate which constitutes what is known as endarteritis. In syphilitic arteritis, therefore, there is both a periarteritis and an endarteritis; the former being usually the primary and most essential process. The endarteritis, however, as it develops gradually produces an occlusion of the arteries. This cuts off the circulation of the blood and leads to softening of the part. There is also a development of a hyaline degeneration in the arteries which some regard as a very essential part of the anatomical change.

Diagnosis.—The diagnosis of nervous syphilis is based upon the history of an infection, the irregularity and fugacity of the symptoms, the intense headaches, the presence of an optic neuritis, the age of the patient, and the results of treatment. In estimating the importance of the history of infection, it should be remembered that the third year after infection is the serious one for the development in particular of those symptoms produced by obliterating arteritis. In hereditary syphilis the presence of the Hutchinson teeth, the hazy cornea, and deafness or other ear trouble help us in diagnosis. The headache of syphilis is rather characteristic. It may attack any part of the head, but is usu-

ally unilateral or irregular, or again it may be bilateral in its distribution. The pain is very intense and sometimes exhibits a certain periodicity. It is usually worse at night. It is apt to last continuously for from five days to three or four weeks. Headache of this character, followed by the paralysis of one or more cranial nerves or by an attack of hemiplegia, is extremely suggestive of syphilis. Optic neuritis is very liable to occur when the disease shows other evidences of being situated at the base of the brain. This optic neuritis is associated with contraction of the visual field, and a characteristic feature of this contraction is that it varies a great deal from week to week. The sex and age of the patient may be taken into consideration in weighing the evidence, and finally the prompt effects of the use of iodide of potassium should have very decided weight.

Prognosis.—It is very difficult to give definite facts regarding the prognosis of syphilis. Unquestionably the outlook is much more favorable than it is for any other organic disease of the nervous system. When the syphilitic process has not produced so much arterial disease as to lead to obliteration of vessels and softening, a very great degree of improvement and even a recov ery may be expected. So far as injuries to the nerves or nerve roots go, we can generally expect a great improvement or cure. Lesions of the convexity are usually amenable to treatment. Syphilitic hemiplegia has a not much better prognosis than hemiplegia from other causes. Syphilitic myelitis has a not very good prognosis, but it is better than that of myelitis due to trauma. Nervous syphilis may last from one to three or four years. The effects of the disease may, if nerve tissue is destroyed, last a lifetime.

Treatment.—As regards the prophylaxis, it is important that persons who have become infected by syphilis should be treated with iodide of potassium in the second as well as the third stage of the disease. After the first year at least, the patient should not neglect to take a certain amount of iodide of potassium four times a year, each course of treatment lasting six weeks. The patient should be warned against indulging in alcohol, against all excesses, mental as well as physical. A laborious life full of worry and anxiety, in which the patient attempts to help himself along with stimulants, is surely provocative of the development of nervous syphilis.

The treatment of the disease when it has appeared consists mainly in the administration of iodide of potassium or sodium. This should be given in beginning doses of ten grains three times a day and increased gradually until the maximum amount which the patient can bear is taken. This maximum is usually between three and four hundred grains a day. In some cases it is im-

portant to give more than this—as much, that is to say, as two hundred grains three times a day, and it is the general experience of American neurologists that results can be obtained by these large doses which cannot be obtained by smaller ones. In my own experience I have known a patient to take five hundred grains three times a day for a considerable time without harm, and indeed with benefit. Usually, however, such extraordinary doses are rarely needed. It is found that, as a rule, patients tolerate large doses of iodide quite as well as smaller ones, and sometimes the iodism produced by small doses disappears when large doses are given. The drug is best administered largely diluted with water or with Vichy or in milk, and taken after meals. Some persons bear it better before meals. It is occasionally advisable to combine mercury with the iodide. This may be given in the form of the bichloride or by an inunction. Other drugs which are of value are the ordinary tonics such as iron, quinine, and the bitters and mineral acids. Plenty of good food, out-door air, and all those things which will improve the general health of the patient are indicated.

CHAPTER XXI.

FUNCTIONAL NERVOUS DISEASES.

FUNCTIONAL nervous diseases are those in which no definite known anatomical change underlies the morbid phenomena. On this account it is customary to classify them on a clinical basis. We can, however, also make etiological and pathogenic subdivisions. Applying such a method now, we have two broadly distinguished classes: the primary, or degenerative, and the secondary, or acquired neuroses. Such a classification is suggestive and helpful, though not perfectly correct, because several factors often enter into the cause of the same neurosis.

- **Primary degenerative neuroses.**
  - Primary neurasthenia, hypochondriasis.
  - Epilepsy.
  - Hysteria major.
  - Hereditary chorea.
  - General spasmodic tics.
  - Myotonia.
- **Acquired neuroses.**
  - From infectious, autocthonous, and mineral poisons.
    - Chorea.
    - Tetanus.
    - Tetany.
    - Rabies.
    - Tremor.
    - Neuralgia.
  - Exhaustion and shock neuroses.
    - Neurasthenia.
    - Hysteria.
    - Exophthalmic goître.
    - Occupation neuroses.
  - Acquired degenerative neuroses.
    - Tic douloureux.
    - Local spasmodic tics.
    - Paralysis agitans.
  - Miscellaneous.
    - Vasomotor, trophic, and sleep disorders.

THE DEGENERATIVE NEUROSES.

EPILEPSY.

Idiopathic epilepsy is a chronic functional disorder characterized by periodical seizures attended with loss of consciousness and usually with convulsions. Mental disturbances may accompany or take the place of the convulsions.

Symptomatic epilepsy is a form in which the periodic convulsive attacks are due to gross organic changes in the brain.

Jacksonian or *partial epilepsy* is a form of symptomatic epilepsy usually, and is characterized by periodic convulsions affecting only certain groups of muscles, and often unattended by loss of consciousness.

Hystero-epilepsy is not epilepsy, but a form of hysteria.

Eclampsia or acute epilepsy is the name given to a single isolated attack of convulsions. It is generally of the symptomatic type.

Reflex epileptiform convulsions are considered to form a distinct type by some (Starr). In this class of cases peripheral nerve irritations produce severe or mild epileptic attacks which are often very frequent in occurrence.

Idiopathic epilepsy shows itself in three rather distinct types of attacks, viz.: that of severe attacks, called the *grand mal;* that of minor attacks, the *petit mal;* and the rarer larvated forms characterized by acute mental disorder and called *psychical epilepsy* or the psychical epileptic equivalent.

Etiology.—Predisposing causes: Heredity is the most potent of any single influence. A history of epilepsy or insanity is found in the family in about one-third of the cases and rather more on the paternal side. Alcoholism and the intermarriage of neurotic persons contribute powerfully to produce the convulsive tendency in children. Powerful emotions during pregnancy, accouchement injuries, and syphilis have some influence. More cases occur in the country than the city, more in temperate climates, and more among in-bred races. All American statistics (Putzel, Hamilton, Hammond, Starr, and myself—186 cases) show a slight preponderance among males. European observers find it the other way.

Age.—The epileptic age is between ten and twenty, and still more definitely between ten to fifteen. In three-quarters of the cases the disease begins before the age of twenty; in one-sixth of my cases before the age of five. After twenty the danger of epilepsy is slight, and when it occurs it is usually due to accidental causes, like syphilis, alcoholism, or plumbism. Idiopathic epilepsy, however, may develop even after sixty. The accompanying table shows graphically the relation of age to the development of epilepsy, chorea, and neuralgias.

Exciting causes. Exciting causes are not present in the majority of cases. The most important are the occurrence of rickets at the time of dentition, fright, injury to the head, sunstroke, infectious diseases, especially scarlatina, masturbation, alcoholism, and syphilis. Masturbation is a real but rare cause, so also is syphilis. The so-called reflex causes are ocular and auditory irri-

tations, worms, dyspeptic states, dental irritations, lesions involving peripheral nerves. Some American observers put much stress on the importance of ocular irritations; European writers have laid more emphasis on disease of the ear. Probably the gastro-intestinal tract and genital organs furnish the most important exciting irritations. True idiopathic epilepsy may be

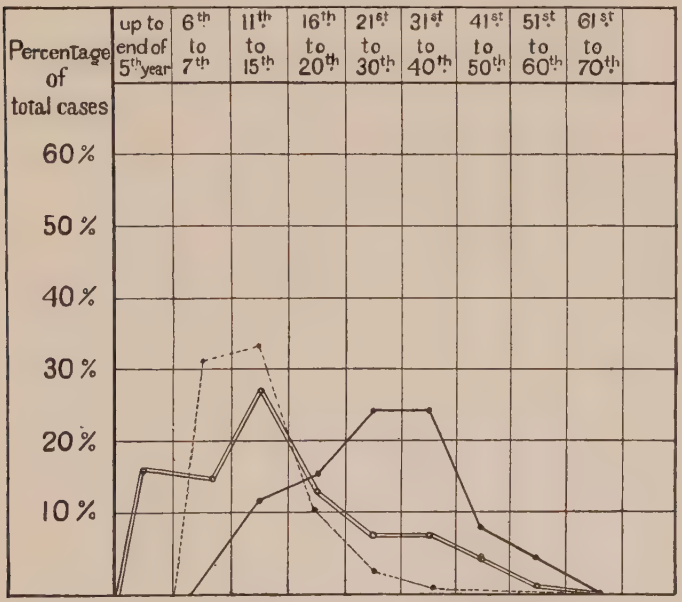


FIG. 189.—TABLE SHOWING PERCENTAGE OF CASES OF EPILEPSY, CHOREA, AND NEURALGIA OCCURRING AT EACH HALF DECADE AND (AFTER 20) EACH DECADE. Double line, epilepsy; dotted line, chorea; single line, neuralgia.

brought out by peripheral irritations; more rarely there occurs only a reflex epileptiform neurosis.

Symptoms of the Convulsion.—The patient often feels some premonitory symptoms for a few hours or a day, consisting of general malaise, irritability, or giddiness. The attack begins in about half the cases with a peculiar sensation called the aura. Often also a loud cry is uttered and the patient falls unconscious to the ground. The face is pale, the eyes are open and turned up and to one side and the pupils dilated. The head is drawn back or to one side, and the whole body is in a state of rigidity or tonic spasm. The arms are slightly drawn out from the trunk, the forearms and wrists flexed, the fingers clinched or flexed in other ways, the legs and feet extended. This tonic stage lasts

for fifteen or twenty seconds; the face becomes congested and then livid from compression of the veins of the neck and stoppage of respiration. Gradually jerky movements of the face and limbs begin and the stage of clonic spasm sets in. The trunk and limbs are now alternately flexed and extended with violent shock-like contractions, the facial and eye muscles twitch, saliva collects in the mouth, and as the tongue is often bitten, it becomes stained with blood. The movements are sometimes so violent that the patient is thrown about the bed or floor, and occasionally a limb is dislocated, usually the shoulder. The urine often, and the fæces occasionally, are passed. The temperature is raised $\frac{1}{2}^{\circ}$ to 1° F., rarely more. The pulse, feeble at first, becomes frequent and tense, and then, as the attack subsides, becomes feeble again. The clonic spasm lasts one-half to one or two minutes. It subsides gradually, and the patient sinks into a stupor, from which he can be roused with difficulty. This stupor is succeeded by a heavy sleep of several hours and a feeling of hebetude which lasts all day. Vomiting sometimes occurs as a terminal symptom. Immediately after the attack there is a temporary exhaustive paralysis, with loss of knee jerk. The pupils contract again and often oscillate. There may be a slight amount of transient albuminuria or glycosuria. The earthy phosphates are found increased; urea is not. There is a distinct lessening of hæmoglobin in the blood (Fèrè) and of hæmatoblasts. Sometimes the attack is followed by others, and for hours the patient passes from one convulsion into another. This condition is called *status epilepticus*. It usually lasts less than twelve hours, but may last for one or more days and until finally death occurs from exhaustion. It only develops in the severer types.

Symptoms of the Minor Attacks.—In the minor attacks (*petit mal*) the patient suddenly stops in anything in which he is engaged, the features become fixed, the eyes open, the face is pale, the pupils dilated, often slight twitching of the facial muscles or of the limbs occurs, and consciousness is lost. In a few seconds the attack is over, and the patient, who does not fall, resumes his work or conversation, being unconscious of what has occurred, except that he has had a "spell." Often there is a warning sensation or aura. This is felt as giddiness, sense of fear, numb sensations of the extremities, flashes of light or blindness, or choking sensations. There may be a cry uttered. The minor attacks are in rarer cases accompanied with sudden forced movements; the patient runs a few steps, or turns round or makes some automatic movements. This is called *procursive epilepsy*.

Symptoms of the Psychical Attacks.—Sometimes the minor attacks are followed by outbursts of maniacal excitement or by sudden violent automatic movements, and in these states the

patient may commit crimes of violence. In rare cases the patient passes into a somnambulic state, during which he performs accustomed acts, such as driving and walking, automatically and naturally (somnambulic epilepsy). This form of epilepsy may come on without a preliminary minor attack, and then it is to be considered a "psychical epileptic equivalent."

Minor attacks may end in convulsions of a co-ordinate type in which the patient jumps, kicks, throws the arms about as in hysterical attacks. These are called hysteroid convulsions.

The seizure may consist of only a short tonic stage and a few twitchings of the limbs, the whole lasting but a few seconds. This is called an *abortive attack*. Under the influence of medication, the severe seizures are often reduced to abortive forms.

Jacksonian or *partial epilepsy* is a form of the disease characterized by convulsive attacks affecting only a single group of muscles or a limb, and generally not accompanied by loss of consciousness. Jacksonian epilepsy is always symptomatic of some focal lesion affecting the cortical motor area of the brain. This may be a tumor, inflammation, or injury. This form of seizure is particularly significant of a slowly growing brain tumor or syphilis.

The *aura* usually consists of a sensation of numbness, prickling, or of a breeze beginning in the hand or leg and passing up to the head, when consciousness is lost. Still oftener there is a peculiar sensation starting in the epigastrium and passing upward. More rarely there are special sense auræ such as flashes of light, noises, or voices, and peculiar tastes or smells. Besides these there occur feelings of giddiness, dreamy states, peculiar sensations in the head, and indescribable general sensations.

The auræ may be divided into

Visceral—epigastric, laryngeal, cardiac.

Cutaneous sensations.

Special senses—flashes of light, etc.

Psychical—emotions, dreamy states, etc.

Cephalic—giddiness, etc.

The aura is thought to indicate the seat of the first discharge of nerve force, and its study is of most importance in connection with symptomatic epilepsies, as will be shown later.

Relative frequency of the different kinds of attacks. The severe attacks are the most frequent, next come combinations of severe and minor attacks, and next minor attacks alone, while the psychical forms are the rarest.

Frequency of the Attacks.—The severe attacks may come on only once or twice a year, and this commonly occurs during the development of the disease. The frequency gradually increases until they occur every month or two or three times a month.

Sometimes the fits occur in groups of four or five every month or two. In very bad cases convulsions occur every day. The *petit mal* attacks are more frequent and usually occur daily.

Time of Attacks.—The moon and the seasons have no influence. More attacks occur during waking hours than during sleep; but two-thirds of the attacks occur between 8 A.M. and 8 P.M. Many patients have their attacks early in the morning just after awaking (*matutinal epilepsy*). Many attacks occur between 3 and 5 A.M., when the temperature of the body and the vital powers are at the lowest. Practically no epileptic attacks occur between 11 A.M. and 12 M. (only 3 in 1,985—Fèrè).

State of Patient Between Attacks.—Epileptic patients often feel better for a time after the convulsion is over. They not rarely suffer from severe neuralgic headaches; the appetite is capricious, often in children it is voracious, but in older cases their may be anorexia; the bowels are usually constipated; the pulse is small, soft, and frequent.

Mental Condition.—A gradual mental deterioration occurs in the great majority of epileptics; but it is slight in some and not very great in others. It shows itself by feebleness of memory, irritability of temper, selfishness, incapacity to concentrate the mind or to carry out a purpose. In children great mischievousness and lack of moral sense, with vicious impulses, may appear. The mental deterioration is dependent on those underlying factors which cause the disease. It is apparently in some cases due to the excessive number of the fits. This is not necessarily the case, nor is it generally true that it occurs more often with *petit mal.* It is more marked in cases beginning very early in life, but this is only true when there are decided marks of physical and mental degeneration present. A certain rather small percentage of epileptics become either demented or insane. True epilepsy is not compatible with extraordinary intellectual endowments. Cæsar, Napoleon, Peter the Great, and other geniuses may have had some symptomatic fits, but not idiopathic epilepsy.

Physical Condition.—Epileptics are rather undersized and of not very robust constitution (Fèrè). They always present some of the marks of degeneration, physical, physiological, or mental. Such marks or stigmata are about ten times more frequent than in healthy persons. The physical stigmata are (Fèrè) short stature, cranial asymmetry (in 71%), short parietal or frontal arc, and triangular skull; in women high prominent forehead; bad teeth badly placed, high palatal arch; facial asymmetry; prominence of occiput and lemurian hypophisis; differences in color, size, position, and shape of pupils; astigmatism (in 75% cases); badly shaped and placed ears; misplaced crown of scalp; low vital capacity; small genitals, atrophic uterus; greater develop-

ment of left side; long fingers. Cranial deformities of pronounced type occur in epileptics associated with idiocy, hemiplegia, and brain defects of early origin. Sometimes, apparently from a premature ossification of sutures, there are the peculiar shapes of the skull known as scaphocephaly, or steeple skull, and plagiocephaly, or obliquely deformed skull.

The physiological marks of deterioration are a lessened muscular strength (as 35 to 50), habit choreas, a rather imperfect eye with excessive amount of astigmatism and functional muscular weakness. There is a lessened vital capacity, weak and slow digestion, and sexual atrophy or irritability. The excretion of phosphoric acid is below normal as compared with urea.

FIG. 190.—DIFFUSE NEUROGLIA SCLEROSIS OF THE CORTEX IN EPILEPSY.

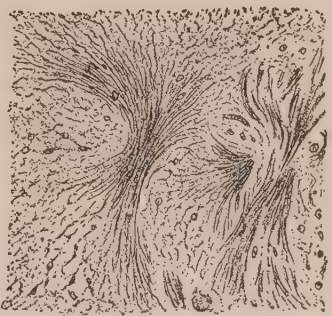


FIG. 191.—SAME, ENLARGED (CHASLIN).

The psychical stigmata are mental feebleness, moral insensibility, irritability, wayward and vicious inpulses, lack of will power, and sexual aberrations.

Pathology.—The body of the epileptic shows sometimes skin eruptions and ulcers, the result of treatment. There are often evidences of local injuries and fractures due to falls. The organs may show vices of conformation. The uterus is frequently infantile or sharply flexed. Deformation of the occipital bone or the atlas so as to produce narrowing of the upper spinal canal has been noticed. The brain may be unduly large or small, but there is nothing constant in this, nor is there an abnormal difference in the weight of the two hemispheres. The convolutions show many anomalies, but there is in them nothing specific. On the whole the convolutional type is a simple one. The pathological change found most constantly in epilepsy is an induration or sclerosis (gliosis). This affects the cornu ammonis rather often (4% to 10%), more rarely the olivary bodies or cerebellum. Besides

this, small patches of induration occur in the gray matter in various parts of the cortex. Chaslin finds a diffuse increase of neuroglia tissue throughout the brain, more marked when the case is older (Fig. 191). Others have found an increase in the neuroglia cells (Kingsbury). In old cases there is often a chronic leptomeningitis, and vascular changes due to the frequent congestions of the brain take place. These consist in varicose and fusiform dilatation of vessels, with evidence of small hemorrhages. Slight degenerative changes in the nerve fibres are also observed.

Bevan Lewis finds in epileptics with insanity a fatty degeneration of the nuclei of the "angular cells" of the second layer of the cortex. In the severer and later stages of the disease this nuclear degeneration is increased so that vacuoles are formed. The cells of the deeper layers are also affected, but to a less extent. The change, though not peculiar to epilepsy, is more extensive and pronounced in this disease.

To sum up: The anatomical basis of idiopathic epilepsy consists in a nuclear degeneration and later a vacuolation of the cortical cells, beginning and most pronounced in the cells of the second layer. Also a proliferation and increase in the neuroglia tissue, this occurring most markedly in various islets or special areas of the cortex. The blood-vessels and connective tissue are involved only secondarily and later.

Physiology.—The epileptic fits are due to sudden discharges of nerve force. The seat of the discharge is the cortex of the brain. The discharging cells are, in the severe seizures, the large motor cells, the function of which is to store up and discharge nerve force. They are under control of the sensory cells (angular cells) of the second layer, which have an inhibitory power. These being diseased their control is weakened and the motor cells "explode" periodically. In sensory and psychical epilepsy the same mechanism exists. The more highly organized cells with large nuclei of the second layer are congenitally or otherwise weak and diseased, the cells below them are not maintained in stable equilibrium and hence periodically break down and "discharge."

The *diagnosis* is based on the character of the attacks and has to be made from hysterical and various toxic and symptomatic convulsions.

The aura, the scream, the quick loss of consciousness, the dilated pupils, the tonic convulsion, the bitten tongue, the emptied bladder, are all characteristic. The hysterical patient sometimes, but rarely, loses consciousness, the epileptic almost always. Hysterical patients do not hurt themselves in falling or bite their tongue, and their muscular movements, while irregular and violent in character, are yet co-ordinate, *i.e.*, they throw themselves

about, kick, strike, etc. Their attacks often are produced by emotion and are ended by some powerful mental or physical impression. The slight rise of temperature in epileptics rarely occurs in hysterics. *Petit mal* and epileptic vertigo are distinguished by the sudden lapse of consciousness and by the sudden pallor and fixation of the eyes, dilatation of the pupils, and slight twitchings of the face. Nocturnal convulsions are usually epileptic.

Eclampsia, or acute symptomatic and reflex convulsions, cannot always be distinguished from epilepsy. The history of the case, the irregular and often prolonged character of the fit, may enable one to make the diagnosis.

Course and Prognosis.—Epilepsy shortens life to some extent; most cases do not live beyond the age of forty or fifty. About ten per cent become demented or insane. Five or ten per cent get well. The remainder reach a certain stage of severity in their disease and continue in it for years. This severity depends on the treatment, the nature of the attacks, and the extent of degeneration which the organism shows. While unquestionably treatment cures or suppresses the disease in some cases, it disappears spontaneously in others. The prognosis of *petit mal* is worse than that of *grand mal;* that of the two combined is worse still, yet not hopeless. The psychical form of epilepsy is the least amenable to treatment. Epileptic insanity and dementia are incurable. Death occurs rarely in the attacks except in terminal stages. Yet the *status epilepticus* is always a source of danger.

Epileptics are said rather frequently to suffer from phthisis. This is, however, a matter of infection and can be prevented. It should be remembered that epileptics who have only a moderate number of attacks, six to fifteen yearly, can get along comfortably for years, doing their work and enjoying a fair share of the duties and pleasures of life. Finally, the following prognostic rules may be laid down: The prognosis is better in males, better if there is a hereditary history, better if the fits are nocturnal or diurnal alone, better in *grand mal*, better if fits occur infrequently, better if they begin after twenty, and better if due to extrinsic causes. The prognosis is very bad in post-hemiplegic epilepsy and epilepsy due to organic disease.

Treatment.—The first and essential rule of treatment is to take cases early and treat them vigorously from the start. Children who have had a few convulsions during the first three to five years of life should be treated as if they might develop epilepsy between the ages of ten and fifteen or earlier. The recurrence of a fit between the ages of five and ten should excite apprehension and call for the most diligent treatment. Another rule is that when epilepsy is recognized in children the case should be

treated constantly for at least three years after all attacks have ceased.

Constitutional Treatment.—Along with the evolution of epilepsy there is probably a progressive diffuse neuroglia sclerosis of the brain. Whether this is primary or secondary, it is at least proper to use those measures which apparently affect this neuroglia proliferation. Nitrate of silver and arsenic and perhaps iodide of potassium are drugs which we have good reason for believing affect this. Hence I advise that all epileptics as the basal medication should be given courses of these drugs.

Besides this, we should use measures that increase vasomotor tone and strengthen and steady the circulation. Nothing does this better than water. Epileptics should be given showers, douches, cold sponge-baths, or wet-packs according to their needs and opportunities. They should also drink water freely. Again, the nervous system is greatly steadied and quieted by mental occupation that interests one. Nothing is more unfortunate than the idleness often enforced on epileptics. I have seen the disease absolutely checked by having a boy learn a trade that he liked.

The next most important indication is diet. In *petit mal* particularly an absolutely non-irritating diet, such as milk, meat, and bread, will quickly lessen or stop the attacks. Meats can be taken in moderation if eaten slowly. As a rule it is a little safer to keep meat out of children's diet for a time; but in adults it is not necessary.

Removal of irritating causes. Malaria if present promotes the convulsive tendency; so also do lead and alcohol; tobacco does not do this, but its use is better stopped, as it is liable to weaken vascular tone and impair digestion. Syphilis causes epilepsy only through producing organic changes.

The rheumatic, gouty, and so-called tubercular diatheses do not stand in any close relation to epilepsy. The condition known as lithæmia, however, in which there is insufficient oxidation and excretion of products of tissue waste, needs attention. Hence the use of bicarbonate of potash, the alkaline mineral waters, and a restricted diet is not rarely indicated.

The importance of reflex irritations has been much overestimated. Still they must be considered. The most serious are those arising from the gastro-intestinal tract, the sexual organs, and the eyes. Phimosis if present must be relieved, and masturbation or sexual excesses stopped if possible. It is admitted now that removal of the ovaries, even if diseased, never cures true epilepsy, though it may help hysterical convulsions.

Astigmatism and hypermetropia should be corrected; also ocular insufficiencies if these are pronounced.

Proper attention to the frequent constipation and dyspepsia is of course necessary. The use of hot water is often serviceable, a glass being sipped slowly before the morning and evening meals. This helps also to carry off the bromides and wash out the system generally. To still further promote this, a purge should be given every fortnight or month. Out-door life and active physical exercise are indorsed by Hippocrates. They do not have any specific influence unless associated with some employment.

Specific treatment. The drugs which have obtained and held a reputation as anti-epileptics are not numerous. They are the bromides, chloral, chloral-amide, belladonna, zinc, nitro-glycerin, antipyrin. Of less value are digitalis, cannabis indica, borax, valerian, and ergot. As adjuvant drugs we have quinine, strychnia, iron, the phosphates, arsenic, silver, the alkalies and iodides. The most valuable of the specific drugs are the bromides.

All bromides act alike in this disease. If one does not cure another will not. Occasionally, changing or mixing reduces the attacks for a time and benefits the stomach. The best bromides are those of potassium, sodium, strontium, ammonium, and hydrogen (hydrobromic acid).

Bromide of potassium is the most trustworthy. Bromide of sodium is more agreeable to the taste, less irritating to the stomach and milder in its effects, but is eventually just as depressing as other forms. Bromide of ammonium has a brief stimulant effect on the circulation. Bromide of strontium has no advantages that I can discover. Bromide of gold is of no use.

Hydrobromic acid is useful in those cases in which there are indigestion and phosphaturia and an alkali is contra-indicated. It produces acne less readily than the alkaline bromides.

Bromides should be given in daily doses of ʒ i., increased gradually until the attacks are suppressed or the dose reaches ʒ iv. to ℥ i. daily. Few patients can tolerate more than this latter dose. Thorough bromidization should be always tried if necessary to stop the fits, and it may be occasionally repeated. But bromidization is sometimes injurious, even making the disease worse, and it must always be employed with caution. When the fits are suppressed the bromides should be reduced, but never entirely stopped for at least two years after the last fit. In most cases, and especially in nocturnal epilepsy, an extra large dose of bromide or bromide and chloral should be given at night. It is very important that the bromides should be chemically pure (most samples are not), that their use should be continued a very long time, and that their depressing effects should be offset by tonics and all possible roborant measures.

Bromides lessen the fits in from eighty to eighty-five per cent of cases. They do no good or do actual harm, as regards fre-

quency of attacks, in from five to ten per cent of cases. Bromides do no actual good to the patient in a much larger proportion of cases.

To prevent bromide acne, arsenic, calcium sulphide, baths, and diuretics are the best measures.

To prevent bromidization, one should adopt all possible roborant measures; use salt-water baths and regular physical exercise, give black coffee, caffeine, cocaine, mineral acids, strychnine, bitter tonics, cod-liver oil. In all cases the patient should dilute the drug, preferably with carbonic-acid water or Vichy, in the proportion of six ounces of water to a scruple of the drug. A few drops of phosphoric acid may be added to this.

The continuous administration of an alkaline bromide in an alkaline water sometimes affects the bladder, and then the bromide can be given dissolved in hydrobromic acid.

The best substitutes for the bromides, when these do no good or do harm, are belladonna, zinc, strychnine, glonoin, borax, and antipyrin.

The best non-specific adjuvants (drugs) to the bromides are potassium iodide (in syphilitic epilepsy), potassium bicarbonate (in lithæmic and rheumatic states), carbonate of ammonium, the hypophosphites, arsenic, iron, and quinine.

The best specific adjuvant to the bromides, as Seguin has shown, is chloral hydrate. By adding five or six grains of this to a mixture the bromide dose can be reduced one-half and the fits still be controlled. Chloral-amide has a similar effect. Both these drugs will sometimes affect the eyes and stomach unfavorably. Children bear nearly as large doses of bromide as adults.

The remedies that are especially useful in *petit mal* are, after the bromides, bromide of camphor, belladonna, glonoin, cannabis indica, cod-liver oil, ergot, counter-irritation at the back of the neck, and cold spinal douches.

For epilepsy in children, besides the bromides it is sometimes advisable to employ milk diet, rest, and oxide of zinc.

In hemiplegic and in Jacksonian epilepsy the actual cautery applied over the scalp is beneficial. For nocturnal epilepsy one may increase the dose of bromide at night and add chloral or digitalis. Strychnine is sometimes useful. Raising the head of the bed or making the patient sleep in a chair at night are measures that may be tried.

For hysterical and erethitic cases, with or in place of bromides give a diet of milk and vegetables, and try turpentine, valerian, or zinc. Belladonna is usually contra-indicated.

Counter-irritation by means of blisters, issues, and setons at the back of the neck is of doubtful value.

For the status epilepticus give large enemata of chloral and

use emetics and purges. Venesection is often efficacious, morphine is dangerous, chloroform is only palliative, and nitrite of amyl is of little value.

To prevent impending attacks the best remedy is nitrite of amyl, which may be carried in a phial filled with cotton. Inhalation of chloroform or ammonia, the internal administration of ammonia, spirits of lavender, or alcohol, a sternutatory, and pressure on the carotids—all are measures which sometimes stop the attack.

Alterative and habit-breaking drugs, such as mercury, iodide of potassium, arsenic, and antimony, are useful in epilepsy, especially in acquired forms due to lead, alcohol, and syphilis. Bromides stop the fits oftener if given early in the disease, if given to young children, and if given in cases that develop after twenty-one.

Injuries to the head which have caused a fracture or a contusion of the brain are the most frequent traumatic irritants. Whenever epilepsy can be distinctly traced to a blow on the head the question of trephining should be brought up. If there is a history of fracture or present evidence of fracture or even evidence of severe head injury, trephining is justifiable. The more marked the evidence of a degenerative constitution and the less marked the evidence of real brain injury, the less hopeful the prognosis. On the whole, surgery can do little for acquired and nothing for idiopathic epilepsy.

CHAPTER XXII.

HYSTERIA.

Hysteria is a chronic functional disorder characterized by nervous crises of an emotional, convulsive, or other nature and by an interparoxysmal state in which certain marks or stigmata are present. Hysteria is essentially a psychosis, and the dominant symptoms are attributable to disorder of the cortical areas of the brain. Its components are the paroxysms, or "crises" as they are called, on the one hand, and the peculiar symptoms of an interparoxysmal state on the other hand. The disease is to be regarded as a definite one, having a certain, as yet unknown, pathological basis underlying it. The use of the word should be much more restricted and definite than has hitherto been the fashion. There are two forms of the disease, hysteria major and hysteria minor.

Etiology.—Of the predisposing causes heredity is the most important. In about seventy-five per cent there is a history of hysteria or some neurosis or psychosis in the parents. The disease is transmitted more often by the mother. Heredity is particularly apt to be important in the hysteria of children; it is a much smaller factor in hysteria of adult males. A hereditary history of rheumatism, gout, and tuberculosis is of very doubtful importance. Hysteria is a disease of early adult life, most cases occurring between the ages of from fifteen to twenty-five in females; it occurs later in males. Hysteria attacks children between the ages of eight and fifteen, chiefly between eleven and fourteen. The disease affects women more than men in the proportion of four to one, varying much with race, climate, and occupation. Hysteria occurs in all classes of life, but rather less frequently in the middle classes than among the poor and the very rich. Male hysteria is more frequent in the poorer classes who are subjected to the exciting influences of alcoholism, poverty, injuries, etc. Hysteria is certainly much less frequent in its severer forms in this country than in some parts of Europe, particularly France. In my experience it is much less frequent than epilepsy in the northern and eastern parts of this country. It occurs, however, quite frequently in the negroes and also in the Latin races of this country. Bad methods of education and bad family training undoubtedly tend to promote the development of the disease.

The most important single exciting factor is powerful emotion, particularly fear. Other emotions of an allied character—excitement, sorrow, anxiety—may bring on attacks. The disease can be developed by imitation. Injuries combined usually with mental shock are fruitful causes of producing hysteria. The infectious fevers, syphilis, diffuse hemorrhages, the poisons—lead, alcohol, mercury, and tobacco—the administration of ether, mental and bodily and sexual excesses are all important agents in developing the disease.

Symptoms.—The symptoms of hysteria are best described under two general heads—first, those of hysteria minor or the hysterical condition, and, second, those of hysteria major.

1. Hysteria minor is characterized by the interparoxysmal condition of emotional weakness, nervousness, hyperæsthesia and pains, and by crises of an emotional character. In hysteria minor there are no permanent objective marks like anæsthesia and paralysis, and no decided convulsive seizures. The patient, who is almost always a girl or young woman, gradually develops an undue sensitiveness, the mind is depressed, and she gets easily alarmed. She has feelings of nervousness and lacks control over the emotions, she laughs and cries very easily and yields to every impulse. She suffers from headaches which are usually vertical and often severe and chronic, and from spinal pains. She sleeps as a rule rather badly and often has disagreeable dreams. She has, under any little excitement, sensations of tickling, fulness or choking in the throat, forming the condition known as globus. Excitement also brings on attacks of trembling or chilly feelings which come and go. There is more rarely a considerable amount of vasomotor instability, as shown by flushings and by coldness of the extremities.

She has with more or less frequency distinct crises of an emotional character, during which she laughs or cries without apparent cause, or at least to an extent beyond her control. She may have attacks of vomiting or headache, or of intense mental excitement amounting almost to delirium. In some cases the patient has somnambulic attacks at night, or she may have under a little excitement attacks of cerebral automatism during which she involuntarily does things that she is entirely unconscious of when she comes out of the attack. The crises are followed by a copious discharge of very light urine. Hysteria minor is closely allied to a condition of neurasthenia or of simple nervousness. It is associated with neurasthenia oftentimes, and is to be distinguished from it chiefly by the peculiar psychical state, the hyperæsthesia, and the crises which have been described. Even in hysteria minor there is a degree of that peculiar mental condition which will be described later and which is known as sug-

gestibility. Hysteria minor is a disease which belongs especially to childhood and early womanhood. It is very liable to become ameliorated and disappear a little later in life or under the influence of proper treatment, but it may continue or pass into the major form.

2. Hysteria major is characterized by interparoxysmal manifestations of anæsthesia, paralyses, contractures, tremors, peculiar mental conditions, and by paroxysms of an emotional, convulsive, or other serious nature. Hysteria major is what is usually meant when one speaks of hysteria; it includes also hystero-epilepsy. The onset may be gradual, but not infrequently it follows some shock, the first symptom being a convulsion, a paralysis, or some emotional outburst.

The *symptoms of the crises* are the most striking and will be described first. The most common of the paroxysms of hysteria are emotional outbursts of crying or laughing; after this come motor disturbances in the shape of convulsions of various types. Besides this we have attacks of severe pain, forming neuralgic crises; attacks of nausea, gastralgia, and vomiting, forming gastric crises; much more rarely there are prolonged attacks of hysterical coughing, hiccoughing, sneezing, or rapid breathing. The hysterical seizure may also take the form of attacks of trance and lethargy, catalepsy, amnesia, and cerebral automatism.

The emotional crises are characterized by appearing without any good cause; the patient laughs without reason, and the laughing continues and is quite beyond her power of control. In the same way, and rather more frequently, crying attacks or attacks of furious anger and excitement come on. Associated with these outbursts there is almost always a peculiar sensation of something in the throat. It is described sometimes as being a ball or pressure or a squeezing sensation. It is called hysterical globus, and is due usually to a paræsthesia of the nerves of the throat and larynx, but occasionally there is also a muscular spasm of those parts. Following the crises there is a profuse discharge of pale, limpid urine.

Hysterical convulsions have two rather well-defined types. One of them is that which comes on also in hysteria minor and is the ordinary form of hysterical convulsions; the other is a much more severe disturbance in every way and is known as a hystero-epileptic or hysteroid attack. In the hysterical convulsion the patient, under the influence of some excitement, injury, or acute gastric disturbance, rather suddenly falls down and begins to go through various irregular movements of the body, such as thrashing with the arms, kicking with the legs, throwing the head from side to side, rolling about on the bed or floor. In the more distinctively convulsive seizure the hands and arms

and fingers are flexed, the legs and feet are extended, the eyes are generally closed, the eyeballs often converged or moved about irregularly, the pupils dilated. There is some lessening of sensation over the body and of the conjunctivæ. The patient often utters noises or screams at intervals. She may bite her lips, but does not bite the tongue, nor does she ever hurt herself in her various contortions. The attack may last for half an hour to several hours, unless some measures are taken to break it up. In other forms of hysterical convulsion there is simply a general shaking or trepidation of the body as though the patient had a chill; in other cases again the main type of movement is that of opisthotonos, the patient rising up upon the head and heels and arching the body as in tetanus. Again the attack may consist simply of a little rigidity of the body, or of a series of rhythmical movements of the head or trunk or limbs, the patient sitting up and oscillating the head or swaying the trunk or moving the arms, uttering at the same time incoherent words. In children the attacks may be associated with peculiar noises and movements in imitation of animals, such as the growling of a dog or the mewing of a cat. This condition is called therio-mimicry. In some instances the attack may be accompanied or may end in a condition of mental excitement approaching delirium. The patients while suffering from these seizures generally appreciate what is going on about them, and will often respond to some stern order for them to cease or will be brought to a state of quietude by pressure upon some part of the body which provokes pain. In women in particular, pressure over the ovaries or epigastrium will abort the attack; the application of cold water or an emetic will do the same.

The Symptoms of the Interparoxysmal State.—Between the crises the patient may be in a fair condition of general health, but usually presents certain definite chronic manifestations of the disease. The most characteristic are sensory symptoms, paralyses, and contractures.

Sensory symptoms. These consist of cutaneous and mucous hyperæsthesia and anæsthesia and anæsthetic disturbances of the special senses. Cutaneous anæsthesia occurs in three forms: the common form is that of hemianæsthesia involving one-half of the body; next in frequency is the segmental anæsthesia involving an arm or a leg or part of the face or head; rarest of all the forms is a disseminated anæsthesia occurring in the form of patches. These various modes of distribution are shown in the accompanying figures. The anæsthesia is a pain anæsthesia chiefly. The tactile and thermic sensations are less markedly affected. The anæsthesia is in some rare cases transferable by means of magnets or electrical irritants or by suggestion. The anæsthesia can also be lessened or removed temporarily by the

application of magnets or coins or pieces of metal. For example, if a silver coin is fastened upon the anæsthetic area, in the course of a few minutes or a few hours there will be a zone of normal sensation under and around the coin. Sometimes the temperature of the skin upon the anæsthetic part is lowered 3° or 4° F., and upon pricking the skin blood does not flow. The anæsthesia is oftener upon the left side in the proportion of three to one. Hysterical anæsthesias are not accompanied with subjective sensations like organic anæsthesias. The skin reflex is usually abolished. Anæsthesias of some kind occur in a very large proportion of chronic forms of hysteria major. They are rare, however, in

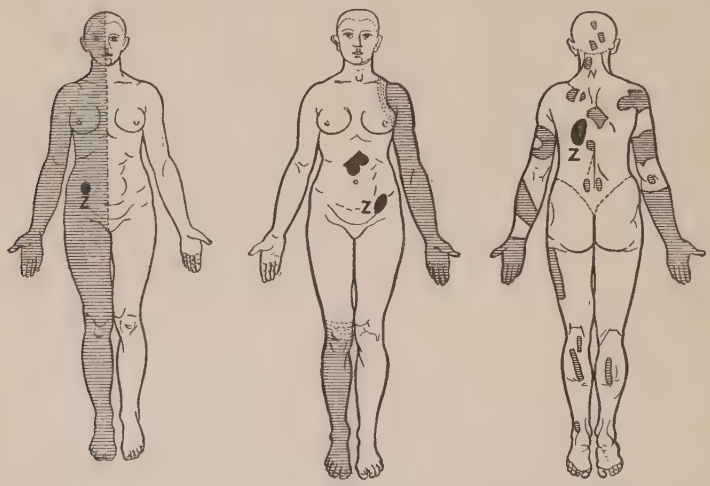


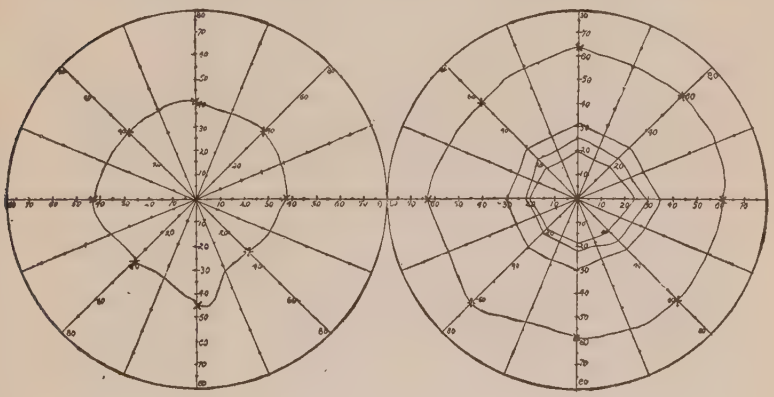
FIG. 192.—THE THREE TYPES OF DISTRIBUTION OF ANÆSTHESIA IN HYSTERIA: HEMIANÆSTHESIA, SEGMENTAL, AND DISSEMINATED. Z, Hysterogenic zones.

children, and are rarer in women than in men in the author's experience. Anæsthesia of the mucous membranes is present chiefly in hemianæsthesia; it then involves the mucous membrane of the mouth and throat, and to a less extent that of the nose and glottis. Hemianæsthesia is usually accompanied with some hemiplegia and often with some tremor. Segmental anæsthesia is also often accompanied with some degree of paralysis of the part.

Visual anæsthesias. One of the most common of the permanent stigmata of hysteria is an anæsthetic condition of the retina. The result of this is the production of a concentric limitation of the visual field and a disturbance in the color sense. Complete loss of this sense may take place or there may be a variation in the way in which the colors are perceived. There may be also

a distinct diminution in the acuity of vision or even a complete loss of sight of one eye. The visual disturbance is most common with hemianæsthesia. It is more marked on the affected side, but exists to some extent on the healthy side. The limitation of the fields is shown in the accompanying cut (Fig. 193).

Hearing. There is sometimes a diminution in the acuity of hearing of one ear, and this occurs, if present, in connection with hemianæsthesia. There may also be a loss of hearing to high and low notes, while hearing is apparently fairly good to notes of medium range; and finally there may be a diminution in hearing by bone conduction, while hearing by aërial conduction is but



O. S. O. D.

FIG. 193.—HYSTERICAL LOSS OF COLOR SENSE AND LIMITATION OF VISUAL FIELD. Color sense absent in left eye and field contracted; in right eye field less contracted; order of appreciation of colors from without in was yellow, violet, blue, red, green (observation by Dr. E. S. Peck).

little impaired, this being due probably to an anæsthesia of the acoustic nerve.

Disturbances of taste in the form of anæsthesia or paræsthesia are quite frequent and are important signs in hysteria. The loss of the sense of taste may only involve the back portion of the tongue and the palate; in other words, the taste field is here limited as it is with hearing and vision.

The sense of smell may be abolished, but this usually occurs in connection with hemianæsthesia.

Hyperæsthesias and neuralgias. Pure neuralgias are somewhat rare in true hysteria, but hyperæsthesia and pains of various kinds are not at all infrequent. Hyperæsthesia occurs in the form of patches at different parts of the body. These sensitive points may, when pressed upon, bring on paroxysms of various

kinds, and they are therefore called the hysterogenic zones (Fig. 192). The most common seat of these zones in women is over the ovaries; in men, in regions correspondng to the ovaries and on the scrotum. Hysterogenic zones, however, may be found just beneath the mammary gland, on the epigastrium, along the spine, and in other places. These zones are sensitive areas; they can be made to disappear by applications of electricity and by refrigeration and counter-irritation. Hysterical patients often suffer from local headaches which are apt to be confined to the top of the head or to the sides near the temples. The pain is severe, sharp, and boring, and may exacerbate with such intensity as to produce symptoms almost resembling meningitis. The spot-like pains are known as hysterical clavus. Hysterical patients occasionally have migraine, facial neuralgia, and intercostal neuralgia. Much more often they have pains along the spine, producing symptoms of spinal irritation. Hysterical patients also have at times attacks of palpitation and pains over the heart, constituting what is known as pseudo-angina. Such troubles are much more frequent in women.

Motor symptoms. The motor symptoms of hysteria are paralyses, amyosthenia, contractures, tremor, and choreic and ataxic movements. The paralyses of hysteria take the form of hemiplegia, paraplegia, and monoplegias. Hysterical hemiplegia occurs usually rather suddenly, often as the result of some severe shock. The left side is more frequently attacked. The arm is most affected, the leg next, while the face is hardly ever involved. The paralysis is not an absolute one, and the patient is able to drag himself along. The deep reflexes are usually not exaggerated and they may be for a short time absent. The paralysis is thus a flaccid one. The gait of the patient is different from that of hemiplegia due to organic disease; in hysterical hemiplegia the patient drags the paralyzed leg after him, in organic hemiplegia the patient swings the paralyzed leg around in a half circle. This peculiarity of the gait, the absence of exaggerated reflexes, the absence of paralysis of the face, and the presence very commonly of other hysterical stigmata are sufficient to enable one to make the diagnosis. Sometimes the face on the affected side is slightly drawn by a spasm so that it appears to be paralyzed, when it really is not (Charcot). Monoplegias affect the arm or leg, very rarely indeed the face, occasionally the eye muscles, and most commonly of all the muscles of the larynx. Hysterical monoplegia is usually accompanied with anæsthesia of the affected part and by other symptoms of hysteria. There are no serious atrophic changes or disturbances of the electrical reactions. Hysterical eye palsies show themselves in the form oftenest of an insufficiency of the internal recti, much more rarely

by a paralysis of the third nerve or some of its branches. In hysterical palsy of the larynx the adductors are involved so that the patient cannot speak aloud, and the condition is called hysterical aphonia. The trouble often comes on suddenly, the patient finding that he cannot speak above a whisper. The paralysis is not so great but that the adductors can be approximated in coughing. The trouble is distinguished from laryngeal inflammation by inspection of the affected part. The abductors of the larynx and the tongue and other muscles of articulation are in very rare cases also involved, and hysteria may produce symptoms resembling a bulbar paralysis. Paraplegia is a rather common form of hysterical palsy; it is usually brought on by emotions of depressing character, often associated with some slight injury. It may be accompanied with a good deal of pain

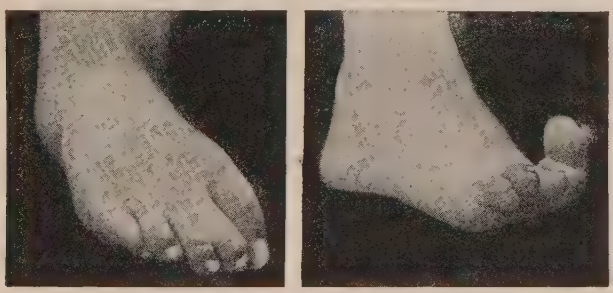


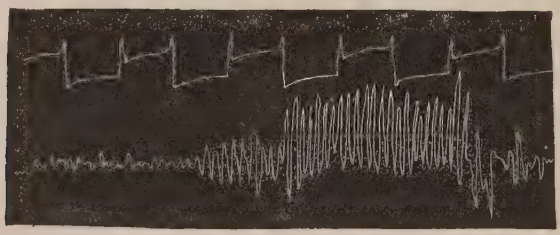
Fig. 194.—Hysterical Contractures.

in the back, and the form of disease which is popularly known as "spinal concussion" consists in many cases of hysterical paraplegia combined with hysterical neuralgia of the spine. In hysterical paraplegia there is very little wasting of the limbs and no change in the electrical reactions. The deep reflexes may be somewhat increased or normal; they are never absent. There is never any prolonged or persistent ankle clonus, but there may be a short or spurious clonus due to a general exaggerated irritability of the nervous system. The sphincters are never involved except temporarily or through some complication.

Amyosthenia is a frequent, peculiar, and interesting symptom occurring in the interparoxysmal stage of hysteria. It consists in a more or less temporary feeling of weakness of an arm or of the legs. Thus a person in lifting a dish from the table suddenly feels the arm give out, and if not careful the dish is dropped; or while walking the patients suddenly feel as though they had lost all power in the lower limbs. This amyosthenic condition is generally temporary, but it may be so permanent as to produce a

certain degree of monoplegia or paraplegia. The amyosthenic condition generally precedes a paralysis. It presents no objective signs in the way of electrical reaction; it involves a whole member, not a single group of muscles; it affects more the anæsthetic side, and when it exists the deep reflexes are usually exaggerated (Tourette).

Contractures. In some forms of hysteria there is a tendency for the muscles to undergo contracture under slight mechanical stimulation such as pressure or a blow. This tendency to contracture in hysteria is called the *contractural diathesis*, and it is an important sign. The contractures may be temporary, disappearing soon after the exciting cause ceases, or they may develop independently and last for a long time. They involve the legs,



REPOSE. MOVEMENT.

FIG. 195.—ILLUSTRATING INTENTIONAL TREMOR IN HYSTERIA (TOURETTE).

arms, and facial muscles, and may be associated with paralysis and anæsthesia.

Tremor occurs in hysteria in a considerable proportion of cases, more especially those in which there is hemiplegia and hemianæsthesia. Hysterical tremor simulates all the various types. The common form is one in which the oscillations occur from five and a half to seven and a half times a second, and it is therefore a tremor of average rhythm. It ceases for a time when the person is quiet or lies in the horizontal position; also during sleep. It affects the head and tongue as well as the extremities, the latter more upon one side than the other. It may be chiefly in the lower limbs. Sometimes it has the type of an intentional tremor, ceasing on rest of the hand and increasing when the hand is moved, as in raising a glass to the lips (Fig. 195). It then resembles exactly the tremor of multiple sclerosis. There may be a slow tremor of four to five and a half oscillations per second; this persists during rest and is but little modified by voluntary movements. It imitates the tremor of paralysis agitans. Finally there may be a rapid tremor of eight to nine oscillations per second. This also persists during repose and is but little modi-

fied by movements. It imitates the tremor of Basedow's disease, alcoholism, and neurasthenia.

The mental state in hysteria is characterized by emotional instability and intense craving for sympathy, weakness of the will, lack of self-control, acuteness of perception, and a constant variability of moods. The hysterical mind is not a dull one, it is not a consciously mendacious or evil one. But there is an abnormal increase of sensibility and an exaggeration of the personal feeling or egotism which leads to selfishness and prompts deceit.

The fundamental defect in the hysterical brain is that it is circumscribed in its associative functions; the field of consciousness is limited just as is the field of vision. The mental activity is confined to personal feelings, which are not regulated by connotation of past experiences, hence they flow over too easily into emotional outbursts or motor paroxysms. The hysterical person cannot think.

Physiologically the condition is explained by supposing that there is a benumbing of the association fibres which normally connect sensory cortical centres with other parts and enable one to compare and adjust new experiences with old. In other words, to reason and form correct judgments.

Underlying the hysterical mental state there is a condition of *suggestibility*, by reason of which ideas and impressions easily become fixed and dominate the mind. The person becomes self-hypnotized and believes he or she is suffering from things which have no objective existence. The pains, palsies, and anæsthesias of hysteria major are pseudo-delusions, differing from delusions of the insane in that the false belief or idea is a subconscious one.

Trophic disorders. In hysterical paralysis a slight amount of atrophy occurs, but only such as would naturally follow disuse of the part. A very few cases have been reported in which a true neurotic atrophy resembling the atrophy that occurs in neuritis or progressive muscular atrophy was present, and it is even affirmed that degenerative reactions may be elicited. Cutaneous eruptions and dystrophies practically do not exist, or, if present, are the result of complicating disorders.

Visceral symptoms. Hysterical patients often suffer from dyspepsia and constipation, also from anorexia and in some cases from persistent vomiting or regurgitation of food. Occasionally the anorexia and vomiting become persistent; the patient refuses food or rejects all that is taken; she emaciates, becomes weak and bed-ridden, and develops into that particular phase of hysteria known as "the fasting girl." In these cases, along with the aversion to food and vomiting, there may be a great deal of gastralgia. The urine in hysteria is apt to be of low specific gravity. Always after hysterical attacks there is a profuse flow of very

light-colored urine having a gravity of only 1,003 to 1,006. Sometimes there is retention of urine; in extremely rare cases there is a condition known as ischuria and anuria, in which for several days extremely small quantities of urine are passed, owing apparently to a suspension of the functions of the kidney. Such cases should always be carefully investigated, to see that the patient does not deceive her attendants in regard to the amount of urine passed.

Vasomotor symptoms are very common. They consist of flushings and pallor, cold extremities, and at times an œdematous condition of one or more extremities. This œdema may be of the ordinary pale, waxy character, pitting upon pressure. In other cases it has a peculiar bluish tinge and it does not pit; the hands, which are the parts generally affected, are several degrees below the normal in temperature, and the limb resembles in some respects the condition in Reynaud's disease. Gangrene, however, never supervenes. This form of œdema is known as the *blue œdema of hysteria.*

There occur in hysteria febrile attacks, and much has been written upon the subject of *hysterical fever.* These so-called hysterical pyrexias may resemble in their course typhoid or malarial fever; as a rule, the temperature runs a very irregular course, and the fever often lasts for weeks or even months. The essentially neurotic origin of these fevers has hardly yet been established, and one can only reach the diagnosis by most carefully excluding all other possible causes.

Anæmia is a very common condition in hysterical patients.

Hystero-Epilepsy.—The form of hysteria which shows itself by the development of severe crises known as hystero-epileptic attacks is extremely rare in this country, at least in its typical phase. It has been particularly studied by the French writers Charcot, Richer, and others. Hystero-epilepsy, as this form of the disease is called, is a true hysteria and not epilepsy at all, nor a mixture of hysteria and epilepsy, though the name would suggest that that was the case. The typical attacks of hystero-epilepsy begin with certain prodromata consisting of a feeling of malaise and irritability which may last for several hours or a day. The attack is ushered in often with an aura, the patient utters a cry, falls to the ground, loses consciousness, and enters into the first phase, known as the epileptoid stage. During this she suffers from tonic and clonic spasms very much like those of true epilepsy. The muscles finally relax, and the patient becomes comatose for a moment and then enters the second stage, that of the contortions and grand movements. In this there is opisthotonos, the body is arched up, and there are violent movements of the trunk and limbs, which undergo flexion and exten-

sion, the movements being all of large range. The next stage is that of emotional attitudes, during which the patient seems to be experiencing intense feelings of anger, joy, or some other violent passion, which she expresses by the postures of her body, the movements of the eyes and facial muscles. This stage over, she enters into the last phase, which is known as that of delirium, during which there is a great deal of mental excitement of a

Fig. 196.—Showing the Different Stages of a Hystero-Epileptic Seizure (after Richer).

depressing character, from which she gradually emerges into her normal condition. To recapitulate: we have in a typical attack, first, prodromata; second, the epileptoid phase, lasting from one to three minutes; third, the phase of contortions and grand movements, one to three minutes; fourth, the emotional phase, lasting from five to fifteen minutes; and, finally, the stage of delirium, lasting a variable time; the whole attack lasting from five to twenty minutes (Fig. 196). In this country we occasionally see hysterical patients exhibiting one or two of these phases,

but very rarely indeed do they ever go through the whole series. The patients who suffer from hystero-epileptic attacks generally during the interparoxysmal stage present many of the stigmata of hysteria such as paralyses, contractures, and anæsthesias.

Hysterical crises which take the form of convulsions or emotional seizures sometimes end or are associated with attacks of *catalepsy* or *trance* or attacks of amnesia and cerebral automatism. As these conditions all occur in other diseases than hysteria, they will be described elsewhere in connection with the subject of the disorders of sleep and of consciousness.

Hysterical persons occasionally are attacked with violent and persistent hiccoughing or sneezing. Sometimes also there come on attacks of extremely rapid breathing or hysterical polypnœa, during which the respirations run up to fifty or seventy a minute. A hysterical cough sometimes occurs; it lasts for a long time. Œsophageal spasm with consequent dysphagia is another one of the somewhat rare phenomena of hysteria.

Pathology.—There is no known anatomical change at the basis of hysteria. We do not find the marks of degeneration as we do in certain forms of insanity and epilepsy. The pathology of hysteria is really its psychology. It is a brain disease affecting primarily the cortex, while secondarily lower centres are involved. The essential trouble is on the one hand a benumbing of the associative and inhibitory tracts of the brain so that the sensations are dammed up till they promiscuously overflow. On the other hand, the receptive areas are over irritable and sensitive. The field of consciousness is narrowed but exalted in intensity.

Diagnosis.—Physicians recognize three different phases of hysteria—a hysterical temperament, hysteria minor, and hysteria major. The hysterical temperament is something with which all women and many men are naturally endowed. It is a condition, not a disease, and does not call for description or elucidation here. Hysteria minor is the hysterical temperament plus certain stigmata and the crises. One should not make the diagnosis of hysteria minor unless he can find these factors. The stigmata we have already enumerated, also the peculiar and varied forms in which the crises show themselves. In hysteria major we have a much greater preponderance of the stigmata and much severer forms of the crises, these being largely of a motor type. Hysteria simulates many organic diseases, and it is often difficult to distinguish surely the real from the spurious thing. The essential characteristics of hysterical forms of disease are the peculiar emotional condition of the patient, the past history of hysterical crises, the presence of the stigmata of hysteria such as anæsthesias, limitation of the visual field, paralyses, and contractures. The variability of the symptoms, their sus-

ceptibility to influence under suggestion and rigorous moral measures, the absence of serious disturbance of nutrition, the sex and age, and the cause should also have weight in guiding us to our decision.

Diagnosis of special forms of hysterical manifestations. Hysterical paralysis is characterized by the fact that there is no marked degree of wasting of the muscles, no electrical reactions of degeneration, the deep reflexes are preserved or exaggerated, and other marks of hysteria are present. Hysterical anæsthesia can generally be lessened over certain areas by the application of the magnet or can be made temporarily to disappear; it is peculiarly distributed in the way described under symptoms and is associated with anæsthesias of the special senses. Hysterical contractures sometimes cease during sleep and always under deep narcosis, and the use of an anæsthetic may clear up the case. They usually follow a fit, an injury, or an operation. They are somewhat increased on attempts to overcome them by force; they are usually associated with paralysis and anæsthesia and other hysterical symptoms.

Hysterical convulsions. These differ from convulsions of epilepsy in the way best indicated by the following table:

Hysterical Convulsion.	*Epileptic Convulsion.*
Brought on by emotion or injury; no aura; no initial cry; movements coordinate; tongue not bitten, and patient never injures herself. Duration perhaps several hours with intermissions; consciousness generally preserved. Micturition and defecation do not occur. No rise of temperature; may be stopped artificially.	The opposite in all these particulars.

The hystero-epileptic attacks are so characteristic that a mistake could not be made.

Prognosis.—The prognosis of hysteria in children is good. They generally get well, although in some cases there is a recurrence later in life. In hysteria minor of young adults the prognosis varies with the severity of the disease and with the physical strength, mental endowment, and social environment of the patient. Mild forms of hysteria under proper treatment usually get well. The severer forms are often intractable even under the best treatment. When a severe form of hysteria occurs in a person of feeble frame who is surrounded with a sympathetic family, the task of rescuing her from her disorder is a very arduous one. Traumatic forms of hysteria which are not infrequently associated with some actual physical injury are often difficult to cure. Hysteria which is associated with some organic disease,

such as a severe pelvic disorder or an organic affection of the central nervous system, has a bad prognosis. Hysteria in the male is generally curable, but it requires vigorous treatment, and spontaneous cure is by no means likely to happen.

Treatment.—The treatment of hysteria may be divided into the mental, mechanical, dietetic, and medicinal.

By all odds the most important factor in the treatment of hysteria is the mental treatment, and the most important measure to be taken is the isolation of the patient. She should be placed where she will not be surrounded by sympathetic friends; where her life will be a regular one; where some occupation may be given which will engross her attention, interest her mind, and call into play her physical activities. In the major forms of hysteria associated with anorexia, emaciation, anæmia, and possibly pelvic disorders, the "rest cure" as elaborated and carried out by Weir Mitchell forms by all odds the most successful means of treatment. In many cases of less severe character a partial rest cure in which the patient is separated from her family but is not placed under such severe restrictions may be all that is needed. In the case of children removal from home is often advisable, and the discipline of well-conducted schools is a most excellent measure.

The mechanical means used in hysteria are hydrotherapy, electricity, massage, and exercise. Of these measures hydrotherapy and electricity take the first rank. In hydrotherapy the douche or jet to the back, the shower and cold plunge, and the half bath are the most efficacious. The technique of their use is given elsewhere. In the electrical treatment the static and faradic currents give the best results. The static sparks often relieve contractures and lessen or remove the anæsthesias, and both forms of electricity seem to have a generally beneficial tonic effect. Massage is of some value in promoting nutrition, and it also has a favorable sedative effect on many cases. Exercise, particularly of an active kind such as stimulates the mind and interests one, is a measure of extreme value and one which has perhaps not been sufficiently appreciated. The use of the bicycle, playing tennis, and horseback-riding are measures which cannot be too strongly recommended to hysterical women; in fact, it is probable that some cases which are submitted to the rest-cure treatment might do better by an entirely opposite kind of procedure.

The drugs which can be recommended in hysteria are not numerous and their power is limited. Valerianate of zinc, turpentine, asafœtida, tincture of sumbul, iron, and the bromides are the most important of the nervines. In hysterical children a capsule containing two grains of valerianate of zinc and one of

sulphate of quinine is often efficacious. Gowers places more reliance upon the oil of turpentine in doses which should be increased to the point of strangury. Pitres recommends the wearing of colored glasses in order to keep off hysterical attacks. Some experimentation is necessary in order to see which color is most suited to the case.

In the treatment of hysterical convulsions the most efficient measure is the administration of an emetic, and this can be best done by giving hypodermically one-twelfth of a grain of apomorphine. Convulsions can be stopped sometimes by throwing water in the face or on the epigastrium; by firm and somewhat long-continued pressure over the ovaries; by the administration of valerian, aromatic spirits of ammonia, or compound spirits of ether.

The Spasmodic Tics (Tic Convulsif).

Spasmodic tic is a disease to which the name of chorea is often, but incorrectly, given. It is a very chronic disorder, and shows itself in the form of quick, electric-like spasms of certain groups of muscles or single muscles. The spasmodic movements are violent, and several rapid contractions succeed each other, after which there is a period of rest. The spasm has a tendency to become localized in certain nerves, especially the facial (*mimic tic*), or even in a single branch or twig, as that to the orbicularis, the zygomaticus, the diaphragm, or the tensor tympani. Spasmodic tic sometimes involves the muscles of expiration and the larynx, and then it has been wrongly called *chorea of the larynx*. Stuttering is a form of *tic*.

The convulsive movements may take a wide range and affect a number of groups of muscles, producing quick, violent movements of the body. They are sometimes accompanied with explosive disturbances of speech. In these cases the patient at the time of the convulsive movement utters some obscene or profane words (*coprolalia*), or involuntarily repeats the last words of the sentence spoken to him (*echolalia*), or spasmodically imitates a gesture made to him (*echokinesis*), or involuntarily exclaims the thought uppermost in his mind, perhaps revealing some secret against his will (*tic de pensée*).

The peculiar disorder of the Maine "jumpers," characterized by sudden violent movements on being touched or startled, is a form of tic. So also are the similar troubles known as *latah*, occurring in Malay, and *myriachit*, occurring in Siberia and Kamchatka.

Most of the special forms of spasmodic tic (mimic tic, wryneck, etc.) have been described elsewhere.

Spasmodic tic with coprolalia affects children between the

ages of six and sixteen years, and by preference the masculine sex. There is almost always a neurotic family history, and the children are nervous.

The disease begins with attacks of violent and irregular movements, affecting generally the head, face, and upper extremities first, then involving the whole body. The movements can be controlled for a time by the will, only to break out with increased violence later. They cease entirely during sleep, which is generally profound.

After having suffered from the disease for a time, the patient will, with the attacks, utter inarticulate cries, or he may begin to repeat or echo the words that he overhears. All this is done automatically and suddenly, with the accompaniment of grimaces and muscular contortions. The special peculiarity of the disease is the sudden interjection by the patient of obscene words and expressions (coprolalia).

The disorder is chronic, lasting for years. It is best treated by isolation, tonics, and ordinary antispasmodics.

Paramyoclonus Multiplex (Myoclonus Multiplex, Convulsive Tremor, Myospasia).

Myoclonus multiplex is a rare disease allied in nature to the convulsive tics and characterized by attacks of quick clonic spasms affecting the trunk and sometimes the extremities also. The disease occurs most often in adult males. It is caused usually by fright, injury, or some violent emotion. Very different forms of spasm have been described under the name myoclonus multiplex, but in the typical disease the patient without warning is seized with sudden and lightning-like contractions of the trunk and hip muscles, which cause his body to be alternately flexed and extended so violently that he is often thrown from the chair or couch on which he is lying. The arms and legs may be also involved, and the attack takes on the characteristics of a general tremor in which the whole body shakes. The facial muscles may be affected. The spasmodic movements are bilateral, although they sometimes begin on one side. The convulsions differ from those of hysteria in that they affect groups of muscles that have not the same physiological function and produce movements which cannot be easily imitated voluntarily. Still this distinction is not a sharp one. The patients are generally neurasthenic or hysterical. They have no paralyses, no anæsthesias, very few pains, and their bodily nutrition is not seriously impaired.

The pathology and pathological anatomy are unknown.

The *diagnosis* is based on the peculiar character of the spasm,

on the fact that the trunk muscles are involved, and that the spasms are bilateral. The disease usually lasts but a few months, but it may extend over a number of years.

The *prognosis* is fairly good.

The *treatment* consists of tonics and the use of chloral. Galvanism seems to be very efficient. Atropine and hyoscine are indicated, and hydrotherapy may also be advantageously employed.

Saltatory Spasm.

This is a curious and rare form of disease characterized by convulsive movements of the legs brought out by touching the feet to the floor. It occurs in both sexes and at all ages, but usually in those of a neurasthenic and hysterical temperament. The phenomena of the disease are exhibited when the patient attempts to stand. The minute that the feet touch the floor violent contractions occur in the muscles of the calves and hips, sometimes in the whole body; these cause the patient to jump, and the movements may be so severe as to throw him down. The seizures are only brought on by the exciting effects of the weight of the body on the feet. Saltatory spasm is probably a form of hysterical spasm. It has been described chiefly by the older writers, and its symptomatology and pathology have not been carefully worked out.

Thomsen's Disease (Myotonia Congenita).

This is a hereditary family disease characterized by the development of tonic cramps when the patient attempts voluntary movements. The disorder is very rare. About forty cases have been reported, and in this country only three, one of them being a patient of the writer's.

Etiology.—Congenital myotony is practically always hereditary and runs in families. It affects males by preference and develops at the time of adolescence.

Symptoms.—The patient notices that on trying to rise or walk his legs are seized with a painless cramp, which in a few seconds relaxes, but comes on again when the muscular movements have been repeated. If he closes his hands tightly a cramp occurs and he cannot relax the grip. If he shuts his eyes he cannot open them for a moment. The muscles of mastication may be affected, but the extremities are the parts most involved. The involuntary muscles are spared. The cramps are increased by cold and nervousness; they are lessened by muscular exercise. The muscles are somewhat hypertrophied, and the patient may present the appearance of a very strong man. The actual strength is fair, but less than would seem. The general health may be good, but the patients sometimes show the signs of low vitality in weak digestion, feeble sexual power, and susceptibility to cold.

The electrical excitability of the nerves is normal, that of the

muscles is increased, and there is produced a contraction tetanus by both currents. In addition Erb describes a peculiar reaction produced by a strong stabile galvanic current. It consists in the appearance of wave-like muscular movements passing from cathode to anode. This was not present in my case or Jacoby's. The mechanical excitability of the muscles is also increased.

Pathology.—The disease is probably a primary muscular dystrophy. There may be, however, a peculiar defect in innervation, resulting from a congenital anomaly of the motor tracts. The muscular fibres are found to be hypertrophied, the striations indistinct, and the nuclei increased.

The *diagnosis* is easily made by the characteristic tonic cramps.

The *prognosis* is bad as regards cure, but the disorder does not shorten life.

Treatment.—Dr. Thomsen, who first described the disease, states that active muscular exercise benefits patients. No specific measures are known.

Congenital Paramyotonia.

Paramyotonia is the name given to a form of myotonia in which the symptoms deviate somewhat from the typical ones that appear in Thomsen's disease. Paramyotonia occurs symptomatically, congenitally, and in a peculiar clinical form known as ataxic. We have, therefore, symptomatic, congenital, and ataxic forms.

Symptomatic paramyotonia is noted most characteristically in a certain form of paralysis agitans. Here the patient, when attempting to walk or to rise from the sitting posture, is suddenly seized with an apparent rigidity of the muscles which prevents him from stirring. The myotonic condition appears also in spastic paralyses of spinal and cerebral origin.

Congenital paramyotonia is a family affection resembling in this respect Thomsen's disease. The muscular rigidity is brought on not by voluntary movements, but by exposure to cold and often very slight degrees of cold. The tonic spasm is a long one and lasts for from a quarter of an hour to several hours. It affects the arms more than the legs. The facial muscles are prone to become rigid. The attacks are followed by some muscular weakness. In congenital paramytonia the trouble is undoubtedly a primary disturbance of the muscles; in other words, a myopathy.

Ataxic paramyotonia is the name given to a disorder characterized by transient spasms like those of Thomsen's disease, associated with distinct ataxia and also with weakness and some anæsthesia (Gowers). This disease is probably located in the spinal cord and should perhaps be considered one of the forms of symptomatic paramyotonia. No special treatment can be given for either of the two latter forms of disease, of which very few examples have been observed.

Akinesia Algera (Pain Palsy).

Akinesia algera is the name given by Moebius to a peculiar form of paralysis which occurs in psychopathic persons and is due to the fact that intense pains are produced by every muscu-

lar movement. The result is that the patient lies helplessly in bed, afraid to stir hand or foot. The disease occurs only in persons who have a very unstable nervous system and generally in those who have a paranoiac tendency. It occurs in adults only. Pain-paralysis comes on gradually and affects eventually all the muscles of the extremities and body. There are no objective disturbances such as atrophy, electrical degenerations, and anæsthesias. The muscles and skin, however, are somewhat tender to the touch. The disease lasts a long time. The patient sometimes improves, in other cases insanity ensues. The disease is essentially a form of insanity, a pathophobia, and is allied to the disorder known as mysophobia. The paralysis is the result of pain hallucination, and the patient is afraid to move the arm or leg on account of this hallucination, just as the mysophobic patient is afraid to touch anything on account of the fear of contamination.

The prognosis is bad, and so far treatment has accomplished little or nothing.

CHAPTER XXIII.

THE ACQUIRED NEUROSES.

Chorea (St. Vitus' Dance).

Under this name various spasmodic disorders have been described. They are to be classed as follows:

I. Common chorea, or Sydenham's chorea.

II. Hereditary chorea, or Huntington's chorea.

III. The convulsive tics.

IV. Hysterical chorea, including so-called chorea major.

V. Various local endemic choreas, such as the electric chorea of Dubini and the electric chorea of Bergeron.

Chorea of Sydenham.

This is the common type of chorea, and is the disease ordinarily meant when the term chorea is used. It is a subacute disorder characterized by irregular jerking and inco-ordinate movements. The disease is a common one, forming about one-fifth of the nervous diseases of children.

Etiology.—Most cases occur between the ages of five and fifteen (see chart, p. 405). It is very rare under five. A few cases occur after twenty, and even up to old age, when a senile chorea is sometimes observed. It affects girls more than boys in the ratio of about 2.5 to 1. In adult life the disproportion is less marked.

It is relatively rare in the negro race, especially in those of pure blood (Mitchell). In this country it is more common in children of German, Hebrew, and Portuguese race.

It occurs in all climates. Most cases develop in the spring months, next in the autumn, next in winter, and last in summer. The seasonal influence varies in different localities. In Philadelphia more cases occur relatively in the spring. In New York there is an almost equal increase in the autumn. School attendance has something to do with these variations. Choreic attacks appear to be related to increase in storms (Lewis). The disease is more frequent in cities, and probably in the poorer classes. Hereditary influence is slight, but it exists. In a small percentage of cases one parent has had chorea, epilepsy, insanity, or a decided neuropathic constitution. A phthisical or a gouty history in parents is also not rare.

The chief exciting causes are injury and fright, mental worry,

and rheumatism. Fright or some emotional disturbance is a cause in about one-fifth of the cases. Acute rheumatism is given as a cause in very varying proportions, ranging from five to twenty-five per cent. In this country it ranges from fifteen to twenty per cent (Sinkler, Starr, Sachs, and personal observations). Endocarditis is developed in the course of chorea in a slightly larger proportion of cases. This may exist without any manifestations of rheumatism. Pregnancy is a cause of chorea generally in primipara and always in young women under twenty-five. Chorea sometimes follows infectious fevers, especially measles, scarlatina, and whooping-cough. It has been caused in rare cases by reflex irritation from an injury, from nasal disease, and sexual disorders. Overstudy and the worry of examinations are factors in causing chorea in predisposed and badly nourished children. Intestinal irritations, such as worms, may excite chorea. Malaria also may aggravate, if it does not produce it. Hamilton describes a form of chorea caused by tobacco-poisoning. Anæmia and malnutrition underlie most cases.

Symptoms.—The disease may begin suddenly, but usually it develops slowly, and it is not till one or two weeks that the symptoms are decidedly prominent. It usually begins with irregular twitching of the hand or face on one side. The child winks, grimaces, and drops things from its hand. The foot and leg become affected later and the child stumbles in walking. In two or three weeks the opposite side is involved, but usually less than the one originally affected. In three or four weeks the disease reaches its height. The patient's movements are then almost continuous. The hands can hardly be used and the child has to be fed and dressed; even walking is awkward and difficult. Speech is indistinct and confused from the irregular movements of the lips and tongue. The muscles of respiration may be involved so that the rhythm is uneven. It is asserted that the heart's action is affected also; but this is unlikely.

The choreic movements usually occur both when the muscles are at rest and during volitional acts. In some cases the disease is chiefly characterized by inco-ordinate movements when purposeful acts are attempted. In other cases voluntary movements can be readily performed, and the muscles twitch only when the limbs are at rest. The movements cease, as a rule, during sleep. But the child sometimes sleeps badly on account of the movements. In severe cases attacks of mental excitement and even delirium come on for several successive nights, and this may be so marked a feature as to form what is called *chorea insaniens*, or *maniacal chorea*. Apart from such phenomena, the mind in chorea is usually dulled, the temper irritable, and the child much harder to manage.

The appetite is poor and capricious, the tongue coated, and the bowels often constipated. The nutrition fails a little; there is anæmia and a tendency to loss of flesh.

The eyes present nothing abnormal. Hypermetropia, astigmatism, and muscular insufficiencies exist, but not much more than in other nervous children.

The child is often worse in the morning and improves toward night. Excitement and physical exertion make the movements worse. There is rarely any pain and never anæsthesia or tenderness. The muscles are weak but not actually paralyzed. The deep reflexes are somewhat lessened and the knee jerk may be abolished. The electrical irritability of the muscles is, as a rule, increased, but there are no qualitative changes. Nocturnal enuresis occasionally occurs. The urine contains an excess of urea and phosphates, and at the height of the attack the specific gravity may be increased.

Forms.—Maniacal chorea is characterized by great mental excitement—especially at night, delirium, with hallucinations and delusions. After one or two weeks the excitement lessens and the patient becomes dull and apathetic. Such cases usually occur in adult women, and they are sometimes fatal.

Paralytic chorea. In this form one arm becomes rather suddenly weak and powerless. A few twitching movements are observed. This form occurs only in children and runs the same course as the spasmodic type.

Chorea of adult life and senile chorea. The disease when it occurs in the second half of life attacks men rather oftener than women; it is not related to rheumatism. There is usually a neurotic family history and even a hereditary history of chorea. The attack is usually caused by emotional disturbances. It runs much the same course as juvenile chorea, but is rather more apt to become chronic. When it occurs in old men it is called *senile chorea.* This type is not to be confounded with hereditary or Huntington's chorea.

Duration—Relapses.—The disease in this country lasts about ten or twelve weeks, ranging, however, from six weeks to six months. There may be great improvement followed by a relapse, and in this remittent manner the disease may last for years. If it lasts more than six months it should be called *chronic.* Relapses occur in about one-third of the cases, and rather oftener in girls. Relapses occur oftenest within a year of the first attack and much oftener in the spring. After three years relapses practically cease. The number of relapses is usually but one, but the disease may recur eight or nine times. Relapses rarely occur in adults except in the chorea of pregnancy.

Pathology.—The seat of the lesions in chorea is the gray mat-

ter of the cortex and its meninges, the pyramidal tract, lenticular nuclei, and the spinal cord. The lesions are in acute cases of the nature of intense hyperæmia, with dilatation of vessels, small hemorrhages, and spots of softening. There is infiltration of the perivascular spaces with round cells and swelling and proliferation of the intima of the small arteries. In chronic cases the evidence of active vascular irritation is less, but there are

FIG. 197.—PERIVASCULAR DILATATIONS IN THE WHITE MATTER OF THE CONVOLUTIONS OF A VERY CHRONIC AND SEVERE CASE OF CHOREA.

perivascular dilatations and increase of connective tissue (Fig. 197). The process suggests a low grade or an initial stage of inflammation. The cause of this is probably either an infective micro-organism or a humoral irritation similar to that causing the rheumatic symptoms and the heart lesions. In a considerable per cent of cases (ninety per cent—Osler), especially in those of long duration, there are fibrinous deposits on the walls of the heart. The hyperæmic process may not be confined to the meninges and motor areas of the brain and cord, but it is only from

the disease in these parts that the symptoms of chorea arise. The presence of points of irritation in the cortex and its meninges and in the deeper parts excites irregular discharges of nerve force and produces the choreic movements. The interruption of the voluntary nerve impulses by diseased foci makes these movements irregular. The apparently special involvement of the lenticular nuclei may explain some of the inco-ordination. In paralytic chorea the pyramidal tract is probably more seriously injured by some single large focus of congestion, exudation, or hemorrhage. Indeed, I have seen a true hemiplegia develop in the midst of an attack. In maniacal chorea the meninges and cortex are more involved.

Diagnosis.—The disease is easily recognized by the peculiar twitching movements. It is necessary only to distinguish the different forms. It must be distinguished from convulsive tic, electric chorea of Dubini, hysterical spasms which include myoclonus, saltatory chorea, and chorea major. The distinctions are not difficult and are given in connection with descriptions of these disorders.

Prognosis.—As regards life the prognosis is very favorable. In this country death from chorea hardly ever occurs in children. It is more fatal in adults. In England the mortality from chorea is about two per cent. Nearly all non-fatal cases eventually get well.

Treatment.—The most important single factor in treatment is rest. The child should not be allowed to take violent exercise or to have any excitement. In most cases he should be taken from school, and in bad cases he should be kept in bed.

Cold sponging or the ether spray daily along the back is useful. Nourishing food and iron are indicated.

As specific remedies, arsenic still heads the list. It should be given in doses of ♏ v. of Fowler's solution t. i. d., increased by one or two drops daily to fifteen or twenty drops or even more. If this causes nausea and gastric pain or headache, the dose should be stopped for a day and then resumed, if possible, where it was left off. Sometimes the sulphide of arsenic is better tolerated than the arsenite. Next to arsenic come antipyrin (gr. v.), antifebrin (gr. iij.), exalgin (gr. iij.), the doses to be carefully increased if needed. Tincture of cimicifuga sometimes helps when arsenic fails. The bromide or valerianate of zinc is also an excellent remedy, especially when there is a hysterical element. The bromides and chloral are useful adjuvants in promoting sleep. Chloral alone is said to be curative if given in doses sufficient to prolong sleep greatly (Bastian). Exalgin given cautiously with iron, in doses increased to fifteen grains a day, is often very useful. Hyoscin hydrobromate in doses of gr. $\frac{1}{100}$ is occasionally

efficacious. Among other drugs of less value are the salicylates, cypropedium, lobeline, physostigmine, and tartar emetic. In chronic and obstinate cases hypodermic injections of Fowler's solution should be tried. Galvanization of the brain and spine is also useful. Change of air sometimes breaks up an attack.

HEREDITARY CHOREA was first described by a Long Island physician, Dr. Waters, in 1842, later by Drs. Gorman and Lyon, and in 1872 by Dr. Huntington. The American cases have been observed chiefly in New York, Connecticut, New Jersey, and Pennsylvania. Cases have been reported also from Germany, France, and England. The disease rarely begins before thirty or after fifty; it occurs about equally in males and females. It is always directly hereditary, either through father or mother, usually the latter. It begins without known cause by twitchings in the face; the movements then extend to the arms and legs. It is attended with progressive mental deterioration, with a tendency to melancholia, and finally ends in dementia. Its course is chronic and usually very slow, lasting ten or twenty years. Post mortem, chronic pachymeningitis and leptomeningitis with degenerative changes in the cortex have been found.

ELECTRIC CHOREA is a name sometimes and wrongly given to very violent forms of ordinary chorea of Sydenham. The term was first applied by Dubini to a peculiar and progressively fatal spasmodic affection which has been observed almost solely in Italy, and which is perhaps of a podagrous or malignant malarial origin. M. Bergeron in 1880 also described an "electric chorea" in which the patients are attacked by sudden rhythmical spasms. This latter disease has a uniformly favorable course. Neither of these diseases resembles true chorea, nor do they have the character of the *tics*.

The term electric chorea, therefore, is one that should be only used, if at all, with a qualifying explanation.

HABIT CHOREA (*Tic Coordiné*).—There are many persons who go through life with some trick of speech, of gesture, or some peculiar grimace. It may be only a shrug of the shoulder, a twitching of the eyes, or a sniff. These various movements are *tics* of the co-ordinate kind. The spasmodic motion is of itself normal, but is inappropriate and misapplied.

Such movements are often seen in children. They sometimes represent abortive attacks of chorea, and sometimes they are the residuum of old attacks. In many cases they are chronic convulsive tics from the start and have little relationship to Sydenham's chorea. The condition is to be treated both by moral and medicinal measures.

Oscillatory spasms and nodding spasms have been described elsewhere.

Procursive Chorea, or Dancing Chorea.—Laycock has described as a separate kind of chorea a rhythmical or trochaic form, which he says affects children, principally girls, and shows itself in spasmodic rhythmical contractions or in sudden rotating or

procursive movements of the body. This has been called chorea procursive, or chorea festinans, by other writers. In many cases it is accompanied with vertigo, when the condition of the patient is similar to that of a person who has been whirling around a number of times. Such cases always have decidedly hysterical characters, although these procursive attacks may complicate ordinary chorea.

Chorea major is a manifestation of hysteria, and will be described under that subject. It is not a chorea at all.

TETANUS.

Tetanus is an acute or subacute infectious disease characterized by violent tonic spasms with remissions and exacerbations. It is called idiopathic when no open wound is found and traumatic when such condition is present. When it attacks infants it is called tetanus neonatorum; when the jaws alone are involved it is called lockjaw, or trismus. A form which affects the face and throat is called head or cephalic tetanus.

Etiology.—It has a special predilection for new-born children in some countries (West Indies) and to a less extent for puerperal women. It affects males more than females. After the first month of life there is practical immunity till after the tenth year. It then increases in frequency up to forty. It is much more frequent in dark races and in some tropical climates (West Indies, South and Central America).

Symptoms.—The disease sets in from five to fifteen days after infection. It begins with feelings of stiffness in the neck and throat and sometimes with chilly feelings. Gradually tonic spasms develop which involve the trunk muscles, causing opisthotonos and other forms of rigid spasm. Trismus, or lockjaw, also occurs. The spasms are attended with intense pain. Sometimes there is a rise of temperature and this may be very high. The disease lasts for two to five weeks. There is evidence of irritation and congestion of the spinal cord and injured nerves, but no special anatomical changes are found. A specific bacillus producing a tetanizing poison has been discovered.

The *diagnosis* is based on the characteristic history and the peculiar spasms. In strychnine-poisoning there is no initial trismus or epigastric pain. In rabies there is also no trismus but a respiratory spasm on attempts to swallow.

The *prognosis* is bad. About eighty per cent of the traumatic and sixty per cent of idiopathic cases die.

The *treatment* consists of complete rest and quiet in a dark room and the administration of chloral, bromide, morphia, and physostigma. Successful results from injection of blood serum of an animal which has had the disease are reported.

Tetany (Tetanilla).

Tetany is a subacute or chronic spasmodic disorder characterized by intermittent or persistent tonic contractions beginning in the extremities and associated with paræsthesiæ and hyperexcitability of the motor and sensory nerves.

Etiology.—The disease is very rare in this country, but relatively common in Europe, especially in Austria. It occurs with frequency during the second, third, and fourth years of life and again at the time of puberty. Its rate of frequency then slowly declines and it is very rare after fifty. It affects males much oftener than females up to the age of twenty, after that the difference disappears. It occurs mostly in the working classes. In infants rickets is often noted. The exciting causes are exhausting influences like diarrhœa, lactation, sepsis, fatigue, mental shock, and fevers; also exposure to cold and wet. Alcoholism, dilatation of the stomach, and intestinal entozoa are also causes. It may be produced artificially by extirpation of the thyroid gland. The disease sometimes appears as an epidemic.

Symptoms.—Tetany begins sometimes suddenly with symmetrical tonic contractions of the hands; at other times there are at first sensations of numbness, prickling or pain in the extremities, with malaise and perhaps nausea; then spasms begin. The attacks affect first and most the upper extremities. The flexors of the forearm and hand are usually involved; the fingers are flexed at the metacarpo-phalangeal joint and extended at the other joints, and the thumb is adducted, producing the "accoucheur's hand." The forearm may be flexed and the upper arm adducted. The knees and feet are extended, the toes flexed, and the foot inverted. In severe cases the muscles of the abdomen, chest, neck, and face are involved. Opisthotonos and dyspnœa may result. The muscles of the face and eyes develop contractions, and trismus sometimes occurs late in the disease. The muscles of the larynx, œsophagus, and bladder may be affected. Fibrillary tremors are observed in the contracted muscles. The attacks are accompanied by paræsthesiæ and cramp-like pains. There may be some abolition of sensation in the skin of the parts affected during attacks. The cramps last from a few minutes to hours or days. They occur during day and night and may wake the patient from sleep. Fever is sometimes present in epidemic cases. The disease has a tendency to recurrence.

While it lasts, both during and between the attacks peculiar phenomena are observed as follows:

1st. *Increased Mechanical Irritability of Motor Nerves.*—The motor nerves show an abnormal irritability, so that on striking the motor point a sharp muscular contraction is brought out. When pressure or a blow is made on the face over or near the exit of the facial nerve from its foramen, contractions of the facial muscles occur, especially those of the lips. This is called the "facial phenomenon." By pressing on the artery and nerve of a limb a tetanic attack can be produced in the muscles supplied. It is probable that it is the pressure on the nerve alone which causes the phenomenon which is called "Trousseau's symptom."

2d. *The electrical irritability* of the muscles and nerves is increased, especially to the galvanic current. Thus a negative-pole closure contraction (CaCC) is brought out by a very weak

current; and if a little stronger, it causes a tonic contraction or cathode closure tetanus (CaCTe) The positive-pole opening contraction (AnOC) may be tetanic, *i.e.*, (AnOTe), and there may be even a cathode opening tetanus (CaOTe), a phenomenon not seen in any other disease. According to Gowers there may be a reversal of the polar formula, so that a positive-pole closure contraction occurs earlier than a negative (AnCC > CaCC). This is certainly rare.

3d. *An increase of irritability of the sensory nerves* is shown by pressing upon them, when sensations of prickling and formication appear along their course. There is an increase also in the electrical sensibility, shown by appreciation of very weak galvanic currents. The auditory nerve reacts to the galvanic current in about fifteen per cent of normal cases, and then only to strong currents and to only a partial extent; but in tetany it reacts in nearly all cases, and with comparatively weak currents (2 to 5 or 6 ma.) on anode closure, anode fixed, and anode opening (AnC Klang, AnDKl, AnOKl) (Chvostek).

The phenomena of hyperexcitability above described vary considerably and rapidly during the course of the disease, and are not always present.

Types of the Disease.—The disease varies in intensity and duration. This variation depends much upon the cause, and there have been made a number of types of the disease based on the etiology. Thus we have

1. Epidemic or rheumatic tetany.
2. Asthenic tetany due to lactation, diarrhœa, exhausting diseases, etc.
3. Thyroid tetany, due to removal of the thyroid gland.
4. Reflex tetany from gastric dilatation and intestinal worms.
5. Latent forms of tetany in which the phenomena of hyperexcitability and paræsthesia occur with very slight if any contractions, and no Trousseau symptom.

Infantile tetany should perhaps be separated from other forms.

Symptomatic tetany from brain disease is also spoken of.

When the spasms are continuous the disease lasts but a few weeks; when they are intermittent it may continue for months. Epidemic cases last but a few weeks. The disease may be said in general to last from a few weeks to a few months. Patients are liable to a recurrence on return of the exciting cause.

Pathology.—The phenomena of the disease indicate a congested and irritative condition of the gray matter of the spinal cord. The cause of this state is evidently in some cases (epidemic tetany) an infectious poison; in other cases mucin in the blood (thyroid tetany), and in other cases it may be a rheumatic or some other toxic influence. Ergot is known to produce symptoms resembling tetany.

In infantile tetany the irritation is apparently cortical and due to meningitis or to rickets and the reflex irritation of disordered bowels. It is doubtful if any reflex influence can be invoked in adults. Tetany is a functional disease and the symptomatic expression of a central irritation. This irritation may be of different kinds, hence tetany has a claim to be called a distinct disease simply on clinical grounds. It has no such definite pathology as chorea or epilepsy. In the very few autopsies which have been made no definite organic lesion has been found.

Diagnosis.—The disease is usually easily recognized by the character of the spasms, their symmetrical nature, their course, and the phenomena of hyperexcitability of the muscles and nerves. Trousseau's symptom is found in no other disease. The "facial phenomenon," the peculiar electrical and mechanical irritability of the muscles and nerves are very rare in other conditions. The sensory irritability and especially that of the acoustic nerve are also characteristic. From tetanus the disease is distinguished by the intermittency of the contractions, their feebler character, the fact that they begin in the extremities and extend to the trunk, and by the absence of trismus at least until late in the disease.

Treatment.—The cause should be removed if possible, lactation stopped, diarrhœa and indigestion corrected, worms expelled, rickets, if present, attended to. Rest, nourishing food, and tonics are indicated. Symptomatically, bromide of potash in doses of ʒ iss. to ʒ ij. daily with chloral furnishes the surest relief. Hyoscine in doses of gr. $\frac{1}{100}$ may be tried. Inhalation of chloroform or injections of morphia are needed in severe cases. In nocturnal tetany Gowers advises digitalis. Lukewarm baths may be of service; so also may ice-bags to the spine. If electricity is used only the weak galvanic current should be employed.

CHAPTER XXIV.

NEURASTHENIA (NERVOUS EXHAUSTION)—NERVOUS WOMEN.

NEURASTHENIA is a morbid condition of the nervous system of which the underlying characteristics are weakness and excessive irritability. The disorder is a functional one. In its typical forms the symptoms are distinctive.

Etiology.—Neurasthenia occurs most often between the ages of eighteen and thirty, next between the ages of thirty and forty, and then between forty and fifty. It rarely, if ever, begins before eighteen or after fifty.*

It affects men and women almost equally in this country. Pure cases are more frequent in men, while atypical forms in which there are complications of hysteria, spinal irritation, and local disorders are more frequent in women.

Americans are, as a race, much affected by it. The cold and dry climates of the temperate zone favor the development of the disorder. So also do high altitudes if they are dry. Hence the climate of this country, as well as its civilization, undoubtedly help to promote the disease. In the West, and especially the Northwest lake region and the Colorado plateau, neurasthenia is said to be rather more prevalent than in the South or East.

It occurs in rural as well as urban populations. The educated classes—professional men, business men, and politicians—furnish a large contingent. Heredity is a very important factor in the primary forms.

The exciting causes are excessive mental strain, anxieties, sudden shocks, injuries, sunstroke, excessive child-bearing, sexual excesses—especially masturbation—irregularities and excesses in eating, drinking, and smoking. Overwork does not cause it, provided there is no great mental worry; and even this can be borne if the patient is regular in eating and sleeping and does not drink or smoke too much. In some cases it follows some single power-

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* Among 100 consecutive cases there were 53 women, 47 men. Ages: fifteen to twenty-five, 25; twenty-six to thirty-five, 38; thirty-six to forty-five, 25; forty-six to fifty-five, 20; fifty-six to sixty-five, 2. There are relatively more cases in the adolescence of man and in the later period of life of women. Nativity: United States, 59; Ireland, 29; Germany, 12; others, 9.

ful physical exertion. Malarial-poisoning, syphilis, and the infective fevers are important agents in causing neurasthenia. So also are lithæmic states, gastric dilatation, and dyspepsia.

Reflex irritations. There are many persons, with delicately balanced organizations, who only require some single depressing or irritating agent to put them in a pathological state. This is the case in those neurasthenics who are made so by reflex causes. Given a neuropathic constitution, neurasthenia may be produced by ovarian and uterine disease. Subinvolution and decided mechanical displacements are more potent in causing neurasthenia than the chronic inflammatory states. Prostatic and urethral disorders, hemorrhoids and fissures, may excite general neurasthenic symptoms. Chronic middle and internal ear disease, causing great tinnitus and partial deafness, often aggravates a neurasthenia, if it does not excite it. Nasal stenosis and hypertrophic catarrh, acting either directly or by disturbing the visual apparatus, may be a cause. Muscular insufficiencies of the eyes, refractive errors, especially hypermetropia and astigmatism, are often important factors in the etiology of neurasthenia, but they are not primary or fundamental. Of reflex influences causing and keeping up neurasthenia, disturbances of the stomach, intestines, and liver are by all odds the most important; next come irritations from the pelvic and generative organs.

Symptoms.—In the neurasthenic condition there are certain common symptoms which will be found in all cases, but these symptoms vary in prominence according to the special setting of the case. The modifying elements are these: (1) age, (2) sex, and (3) neuropathic constitution.

In ordinary cases the patient after some years of arduous work, anxiety, and irregular habits, during which he has kept himself continually on a strain, begins to have feelings of nervousness, restlessness, and irritability which are new to him. He has trouble in sleeping and wakes unrefreshed: he loses interest in his work, which tires him greatly. His appetite is poor and his digestion disordered; he has vertigoes and peculiar feelings about the head; and he begins to get alarmed about himself. The symptoms progress until he finds he can no longer work, he is irritable, depressed, and discouraged, and finally begins to take himself in hand. In some cases, after a sudden fright or injury and shock, all these symptoms develop rapidly.

In younger persons the history is somewhat different: the patient has perhaps indulged in sexual abuse; then suddenly learning that it is a very dangerous practice he experiences a fright. He becomes nervous, worried, and mentally depressed. He too has cerebral paræsthesiæ and insomnia: he loses interest in his studies, cannot concentrate his mind, broods over his early indiscre-

tions, and is panic-stricken over every nocturnal emission. Getting some comforting assurances and advice from his physician, he improves and perhaps gets well, but not infrequently has for several years a train of disturbing neurasthenic symptoms. As in older cases, the neurasthenia may come on suddenly from shock, violent physical exertion, and over-use of tobacco.

Symptoms in Detail.—The neurasthenic patient suffers from a peculiar feeling of *nervousness*, discomfort, uneasiness, and sense of unrest; or from feelings of tension, as though under a stimulant. He loses interest in his work, *dreads to assume any responsibility*, has morbid fears regarding his business, his sexual function, his general health, his future, or some trivial subject. Some idea fixes itself upon him and he cannot rid himself of it. He has difficulty in concentrating his mind on work. Periods of depression attack him. These mental states vary much, and may be so paroxysmal as to form "crises." His sleep is poor, disturbed by dreams, *and he awakes unrefreshed;* sometimes there is a morbid somnolence. Slight attacks of vertigo annoy him.

The sensory symptoms consist of feelings of pressure on the top of the head, as of a hand or cap pressing on the scalp (*the helmet symptom*); pain or weariness in the back of the neck; *numbness of the hands* or feet, creeping sensations up the back or limbs, burning feelings in the ears, pricking of the tongue, a sense of oppression of the chest and of "sinking" in the epigastrium. These form what are termed *neurasthenic parœsthesiæ.* Neurasthenics in about half the cases suffer from headaches. These are diffuse and frontal or occipital. The pain is persistent, worse after exertion, and not very intense. A boring occipital pain, *neckache, and neck weariness* are very common, and women often have severe pains along the spine. Intercostal neuralgia, especially on the left side, is common. General neuralgia, aching of all the limbs, feeling of soreness of the scalp or of the muscles, as though one had been beaten, are often complained of.*

The Special Senses.—Neurasthenia does not, as a rule, cause very serious visual troubles. There is always some weakness and increased irritability of eyesight. If the patient uses the eyes much these symptoms may become very marked. There is a feeling of pressure and discomfort or pain in the eyeballs. Headache comes on. Vision gets blurred; there are spots before the eyes; excessive light is annoying and moderate light dazzles. Reading soon tires and causes pain; the lids twitch; the eyes easily redden and water. Examination shows in early life mo-

* Neurasthenics with a hypochondriacal tendency sometimes suffer from a persistent delusional pain located in a certain region, such as the arm or back. This condition has been called *topoalgia.*

bile and often dilated pupils; they are rarely uneven; there is a slight lessening of visual acuity. The visual field is normal, but sensation in its periphery tires very easily, so that after long test ing there is apparently a concentric limitation of the field. The color sense is unaffected. Muscular insufficiencies, especially of the internal recti, often occur. The patient's visual memories are lessened, so that the neurasthenic cannot remember faces and places as he has been accustomed to do. If the patient has any refractive error, especially hypermetropia or astigmatism, his eye symptoms are more marked and cause him so much annoyance that the general symptoms get worse. The eye trouble may thus keep up the neurasthenia, but it will not alone cause it, and treatment of the general and local symptoms by attention to the eyes alone is irrational and disappointing.

Neurasthenics are often over-sensitive to noise. This is usually simply due to the general mental irritability; but sometimes there is a distinct hyperacousia, and slight noises or even agreeable music drive the patient nearly wild.

Subjective disturbances of taste occur in cases complicated with digestive troubles.

There are no peculiarities regarding the sense of smell other than a general over-sensitiveness to odors.

The general muscular strength is lessened, and the patient very soon tires, or even completely collapses, after slight exertion. Muscular effort or mental excitement causes a *tremor of the hands;* often this is a persistent symptom. This tremor is "fine" and vibratory in character (about twelve to the second). The skin reflexes and tendon reflexes are exaggerated. There is a slight amount of "static ataxia," *i.e.*, the patient sways more when standing erect with closed eyes than when his eyes are open, the reverse being the case in healthy people. The eyelids quiver when tightly shut. The general nutrition is not usually much affected; if anything, the patient loses weight.

There is decided evidence of loss of vasomotor tone. This is shown in the *cold hands and feet*, tendency to hyperidrosis of the palms and soles, flushings and pallor of the face, sense of fulness in the head, variations in the specific gravity and amount of the urine. Palpations of the heart easily occur, and examination of the pulse shows it to have *frequent and rapid variations in tension*, there being generally abnormal lowering. The vasomotor reaction produced by drawing a blunt instrument over the skin is more pronounced than in healthy people. A deep red line appears after pressure, which lasts twenty or thirty minutes. In rare cases an artificial urticaria can be produced. The phenomenon is called "dermography."

The sexual functions are weakened and irritable, and inter-

course is imperfect and exhausting. In the younger and more neuropathic cases emissions occur, and morbid fears regarding this function annoy and depress the patient. These fears and introspections occupy the mind and furnish to the patient the belief that all his troubles hinge upon his early indiscretions or his present rather frequent emissions. Sometimes there is, in fact, such an extreme degree of seminal loss as to keep up the neurasthenic state. The genitals become relaxed, there is a slight discharge of urethral and prostatic secretion, especially after stools. In women dysmenorrhœa, menorrhagia, and slight irregularities in menstruation occur.

The digestive organs, including the metabolic tissues of the liver, are almost always at fault. There are a great many nervous affections of the stomach, such as gastralgia, gastroxynis, muscular atony, etc. These disorders may be associated with neurasthenia and be the cause of part of the trouble. The gastric trouble in rare cases may even be primary, or hold equal rank with the nerves in the symptom complex. Such, however, is by no means the rule. The nervous asthenia leads to the gastric dyspepsia, and this again makes the neurasthenia worse. The special symptoms caused by the stomach complication are a sense of congestion and fulness in the head, dizziness, headache, palpitation, and abdominal pulsation; belching, feeling of pressure and fulness in the epigastrium, nausea, pyrosis, anorexia, and alterations of taste.

The hepatic functions are weakened and slowed down; and a *neurasthenic liver* is one of the common and fundamental conditions of the neurasthenic state. This produces intestinal dyspepsia, fermentation, and constipation. The blood absorbs an excess of ptomaïnes and imperfectly oxidized products, causing many of the general nervous symptoms already described, such as the paræsthesiæ, dizziness, somnolence, and head pressure. This condition is called "*lithæmia*," or nervous gout. It is not so much a distinct diathesis, like the gouty or rheumatic, as it is a morbid condition from accumulation of metabolic products.

The intestinal movements are sluggish, and the patient suffers from flatulence and feelings of distention. Sometimes there is a nervous diarrhœa alternating with constipation.*

Sometimes the striking symptom in a neurasthenic patient is the disturbance in the heart's action. This shows itself in feeble-

* *Enteroptosis and Neurasthenia.*—A form of neurasthenia has been described by Glennard under the head of enteroptosis, or prolapse of the abdominal viscera. This occurs in women who have lax abdominal walls, with some descent of the stomach and intestines, and stretching of the suspending ligaments and mesentery. In these cases there is usually some dilatation of the stomach, atony of the intestinal walls, and consequent symptoms of indigestion.

ness of the heart beat and heart sounds and smallness of the pulse. Palpitation occurs on slight exertion, and there may be pseudo-anginal attacks, or sensations of oppression and discomfort over the heart. The patient also complains of feeling the beating of the heart, perhaps all over the body. The pulse may be very slow, especially in the morning. General neurasthenic symptoms are present. Patients with heart disease are liable to be especially annoyed by various evidences of heart weakness and irregular action.

The respirations are normal in number, but shallow, and *deficient respiratory expansion* may be noted. A feeling of pressure on the chest is often described.

The younger patients and women are generally anæmic, but in middle-aged adults this is not so; the face is full and ruddy and the mucous membranes of good color.

The urine in the young and neuropathic cases is variable in specific gravity, but generally low, 1.012 to 1.018; under excitement it may drop to 1.002. Phosphates, earthy and alkaline, are excreted in excess, while the nitrogenous solids are often diminished in amount. In older cases with more digestive and hepatic disturbance and lithæmia the urine is heavy, 1.024 to 1.036, quite acid, and contains at times excess of phosphates and urates. Oxalates are also found, but indicate no more than does excess of urates, *i.e.*, a lithæmic state. The amount of urine passed is variable. At times there is polyuria, but on the whole the total amount is below normal.

The Modified Types.—*Neurasthenia in Adolescence—Degenerative Neurasthenia.*—When neurasthenia begins under the age of twenty-five, there is a more distinct evidence of neuropathic constitution. The mental symptoms are more marked and approach those of hypochondriasis. There is greater depression and greater introspection. Morbid sexual ideas, fear of impotence, remorse over past indiscretions in venery; spermatophobia; morbid fears of contamination, of being forever incapacitated for procreation or sound mental and physical work, annoy the patient. The general nutrition is affected. He is anæmic, has cold extremities, clammy hands, dilated pupils. His urine is of low specific gravity and contains an excess of phosphates, both earthy and alkaline. Pains, paræsthesiæ, vertigo, head pressure, and gastric disturbances are not so marked.

This type of neurasthenia is often described as "sexual" or as "primary." It is really a manifestation of neurotic degeneracy, and may be looked upon as an abortive form of paranoia. When more fully developed the degeneracy shows itself by such symptoms as fixed ideas, and morbid fears such as agoraphobia (fear of open places); claustrophobia (fear of closed places); mysophobia

(fear of contamination); pathophobia (fear of disease); anthropophobia (fear of society). Abortive forms of *folie du doute*, or the doubtness-madness, are also present in this type.

Neurasthenia of Women in Adolescence.—Young nervous women are usually anæmic, dyspeptic, and constipated; their nutrition is not good; they are thin; they excrete large quantities of limpid urine; they often have spinal pains and headaches of the migrainous and other types, and almost all of them suffer more or less at the time of menstruation. They have mobile pupils. Their special senses are keen, to the point of hyperæsthesia. The heart is irritable; the vasomotor centres are unstable. They have cold, clammy extremities. They are vivacious, quick, observant, impatient, living on their "nerves," fond of excitement, and indulging in amusements, for which they pay the penalty by prolonged exhaustion.

Neurasthenia of Women in Early and Middle Life.—It is the nervous women between the ages of twenty-five years and forty who make up the majority of female neurasthenics. They are, so to speak, the wreckage of the young nervous women and of healthy young women for whom the burden of life and domesticity was too heavy. For they are usually married and are sometimes prolific mothers, though the worst cases are the childless or the "monopædic." The principal symptoms from which they suffer are nervousness, weakness, insomnia, dyspepsia, morbid and depressing ideas, headaches and neuralgias, vertigo, tremor, and vasomotor symptoms.

They often have migraine and backaches, but they do not have fully developed spinal irritation; they suffer from peculiar feelings of numbness in different parts of the body; they often exhibit a marked tremulousness of the hands, while the cold extremities and wet hands are not so commonly observed. They become easily exhausted. They are excessively nervous and irritable; slight noises will startle them; they cannot read long without getting a pain in the head; they suffer from time to time from attacks of profound mental depression, and often have fixed ideas of a melancholy type; they have, at times, "crises," or "nervous storms," which may take the form of a headache, a fit of blues, or a lively domestic disturbance. The dyspepsia they have is not of a very severe type; it is usually accompanied with fermentation and acid eructations. The urine always varies in quantity and specific gravity. Sometimes they will pass large amounts of limpid urine. Such women are often called hysterical, but the somatic and asthenic symptoms are more prominent than the psychical, and they do not have hysterical stigmata.

Climacteric Neurasthenia.—At the climacteric in women, and sometimes at a corresponding age in men, neurasthenic symptoms

develop. In women these symptoms are mostly of a vasomotor character. Hot flashes, flushing, nervous irritability, poor sleep or morbid somnolence, pains in the back and head, sensations of fainting and sinking, tendency to mental depression, are among the chief symptoms in women, among whom this trouble may be regarded as a climacteric neurasthenia. Evidences of vascular tension and of beginning arterial degeneration will be found. In men the symptoms do not greatly differ from those of the ordinary type. Both in men and women this form of neurasthenia runs a comparatively short and favorable course.

Course and Duration.—Neurasthenia is a chronic disorder and lasts from one to five or ten years, depending largely upon treatment and habits of life. The forms occurring at the climacteric are the shortest and most favorable, those occurring in early middle life come next, while those forms which develop in youth, if dependent upon neurotic constitution, are the most chronic. The disease lasts longer in women than in men.

Pathology.—Neurasthenia is a condition in which the nutrition of the nerve cells is primarily at fault. These cells have lost their power of building themselves up into stable compounds. They easily break down under slight irritation, and send out consequently but feeble impulses. These facts are indicated by the terms "irritability" and "weakness." It is thought by some that this dystrophic condition especially involves those parts which make up the vasomotor neuro-mechanism; and if this be so, it is the nervous tissue of the medulla and central parts of the spinal gray matter that are the most involved. There is little doubt, however, that the cortical gray matter and higher centres of the brain are also implicated, for the mental symptoms are always prominent. It is certain that the peripheral nerves are not primarily at fault. Since it has been shown that the sympathetic system is only an outflow from the cerebro-spinal, and that many of its ganglia are rudimentary and functionless, the idea that neurasthenia is located in the "ganglia" of this system becomes quite untenable. The view that neurasthenia is usually lithæmia and of gastro-hepatic origin I have tested thoroughly. Patients rarely get any better by treatment based on this hypothesis.

Vascular changes in the nervous system take place. These are of the nature of cerebral hyperæmia, and probably hyperæmia of the spinal gray matter. More rarely there is anæmia.

The importance and dominance of the vasomotor changes in the skin and internal viscera have been already alluded to under the head of symptoms. This vasomotor disturbance is one characterized by a weakness of the inhibitory vasoconstrictor centres. As a consequence the vessels dilate and contract irregularly in

the different systems of the body. Furthermore, they do not respond normally to the stimulus of functioning. When brain or stomach acts it calls for a definite and well-regulated supply of blood; in neurasthenia it does not get it, and hence the morbid symptoms.

The nervous system controls the metabolic processes of the body, which are especially active in the liver, spleen, and muscles. In neurasthenia this process is disturbed, and the liver particularly is atonic or sluggish. This disturbance has already been described under the head of digestion.

In other cases metabolism is affected differently; it seems to be simply too slow. The tissues do not build up and break down at a normally rapid rate. The urine is of low specific gravity and the total solids excreted are less than normal.

One exception to this slowness of metabolism occurs. In all forms of neurasthenia there is a tendency to excessive breaking up and discharge of the phosphates, both alkaline and earthy.

Diagnosis.—Neurasthenia must be differentiated from simple nervousness, degenerative forms of insanity, such in particular as paranoia, from hypochondriasis, from melancholia, hysteria, and spinal irritation.

Nervousness may be a natural characteristic, a part of the temperament. As long as the patient does not suffer and is not incapacitated from work and enjoyment, we can somewhat arbitrarily say it is not neurasthenia.

Peculiar sexual perversions, doubting mania, pronounced forms of morbid fears, may be associated with some neurasthenic symptoms, but the diseases mentioned are essentially psychical disorders and dependent on a congenitally deficient brain. The hereditary history is usually bad. The clearness and dominance of the mental symptoms are sufficient for a diagnosis. The same remarks apply to *hypochondriasis.* The mental depression and fixed ideas are altogether disproportionate to the other symptoms.

In melancholia there are pallor, anæmia, loss of flesh, anorexia, insomnia, coated tongue, and constipation, often a lowering of temperature, a profound and constant mental depression. This must often be looked out for in women at the time of the climacteric. The mild types of melancholia are found oftener in women.

Hysteria may complicate neurasthenia. It is found much oftener in women, but in the neuroses following shocks and injuries it is seen in men. Hysteria is essentially a mental disease involving the emotional faculties and the will, while the bodily condition may be good. It is characterized by various crises of a convulsive or emotional character. There are certain stigmata

almost always present which enable one to recognize it. These are the anæsthesiæ of the special and general senses.

Spinal irritation is usually a complication of neurasthenia or hysteria, and presents easily distinguishable characters. It may occur quite independently of the distinctive symptoms of the neurasthenic state.

The *prognosis* is in general favorable. Most patients get well, or practically well, so that with care they can enjoy life and attend to its duties. But the patient almost always has some reminders that he cannot overtax himself or do quite as much as can a person of strong nerves. This is especially true up to the age of forty-five or fifty. The lessened sensibility of the nerves in the declining years then comes to his help; he gets the benefit of his past years of regularity and temperance; his late years are his best, and he lives to enjoy many of them. Neurasthenics, if they get well, get old. Neurasthenia does not lead to insanity or epilepsy or organic disease; but it may be supplanted for a time by an asthma or severe neuralgia or other neurosis.

Treatment.—It is of the first importance for the physician to get a firm control of the patient. This can only be done by making the most careful and scrutinizing examination, and by so acting as to make the patient feel that he understands his case and is master of its character. The patient should then have his course of treatment laid out for him. If he does not follow it strictly all treatment should be stopped. Women are harder to manage at their homes than men, and it is better to isolate them somewhere if possible.

Rest or lessening of business and domestic duties is fundamental. Travelling is bad, but the patient can be sent to stay at such places as the Azores, West Indies, Bermuda, or to some special point in Southern Europe. A trip along our great lakes, camping out, or long sea voyages in rather warm climates are to be advised.

The diet should be rather nitrogenous than otherwise, but fats and green vegetables and certain fruits may be added. Sugars and starches are to be avoided, though malt preparations usually agree. The nutrition of the patient cannot be too carefully watched. He should be weighed and sometimes fattened, using water, malt, oil, and cream for this purpose. The amount and specific gravity of urine must be noted, and his diet regulated accordingly. Neurasthenic patients usually drink too little water or else too much at meal-time. Water should be drunk between meals or half an hour to an hour before meals. Neurasthenics need to be properly fed, but not to be stuffed. In many cases a low diet, but one perfectly digestible, is most indicated. Meals should be taken at fixed hours.

A cold sponge bath, or a shower or a strong jet of cold water

on the back, or a cold plunge, are measures that are almost invariably indicated. In irritable cases the wet pack for three-quarters of an hour, followed by the cool wash-off, has a very quieting effect. A lukewarm bath at 95° to 98° at night for half an hour relieves the paræsthesiæ and insomnia. The skin should have a thorough rubbing every day.

Respiratory exercises for expanding the lungs, light exercise, such as swinging clubs or dumb-bells, throwing balls, swinging from horizontal bars or rings, bicycling, horseback-riding, tennis, rowing, fishing, etc., should be encouraged. The exercise should not be severe; it should be interesting, it should be done in fresh air, and it should bring into play the lungs and arms more than the legs. Walking does little good, though it is better than nothing.

The drugs of most use in neurasthenia are the bromides, nux vomica, mineral acids, quinine, iron, valerian, the hypnotics, and saline and alkaline laxatives. Many drugs of repute, such as arsenic, phosphorus, zinc, cannabis indica, belladonna, are of no especial value.

The bromide of sodium or potassium should be given in doses of fifteen or twenty grains three times daily; it should be kept up for only a week and then gradually reduced to one-half dose once daily. At the same time the patient may be given a tonic mixture containing quinia, strychnia, and, if indicated, iron. Quinine must be given carefully, as it causes increase of nervousness in many.

Phosphoric and muriatic acids are the two mineral acids most often of use. These acids are usually better given after meals. In mild cases a tonic and sedative may be combined by giving a mixture containing hydrobromic acid with nux vomica and pepsin. As a matter of convenience the bromide of lithium in five-grain doses may be put up in capsules with hydrobromic acid and glycerin. Tincture of nux vomica sometimes acts better than strychnia, but it must be given in large doses—℥ x. increased to ℥ xxv. or ℥ xxx. The saccharated carbonate or albuminate of iron, or Blaud's pills, if given, should be given generously, *i.e.*, in doses of thirty grains daily.

The insomnia, if present, yields usually to the bromides, but will return unless further treatment is given. After using bromides for a time, therefore, it is better to substitute half a drachm of sulphonal or of chloralamide, or gr. xv. of chloral, or a drachm of paraldehyde. Lukewarm baths (95° to 100°) may be used as adjuvants. The dose of the hypnotic should be gradually lessened and not suddenly and irregularly stopped.

If the patient is lithæmic or arthritic, he should be made to drink an alkaline water, like the lithia waters or Carlsbad, and his bowels are better kept open by means of salines than pills.

Antipyrin, potassium bicarbonate, and the salicylates often help painful symptoms in such cases.

In those forms of nervousness in which there is a great deal of melancholy quinine is of use, and when quinine cannot be borne in ordinary doses the patients will often tolerate Warburg's tincture.

In cases in which there is a good deal of cerebral hyperæmia and occipital pain, aconitia and cupping or burning at the back of the neck are indicated.

In young patients with seminal troubles, atropia, camphor, and turpentine may be added to quieting mixtures at night. In those with an hysterical element, five to ten grains of valerianate of zinc should be given daily for three or four weeks.

Young nervous women require tonics, such as Warburg's tincture and large doses of Blaud's pills, together with systematic cold applications to the spine; and, for certain periods, the bromides, valerian, and nervines of that character. Marriage may help them; but, unfortunately, these women are sometimes very prolific. The cultivation of athletics would be the greatest boon that young women could receive. All girls should be examined, shown what deficiencies exist, and taught to make them up by gymnastic exercise.

In neurasthenic women with painful backs or with a hysterical complication, much can be said for electricity. General and spinal galvanization and the static sparks are the specially efficient forms. General electrization is a decided mechanical tonic and useful in almost all forms of neurasthenia; but it is more useful in cases in early life, in women, in the asthenic, and in those with spinal pains. Electrical treatment should be given three or four times weekly for about six weeks, then suspended.

Massage has no special or curative value in neurasthenia, except as part of the rest-cure. Mildly used it soothes and quiets many patients and has a symptomatic use. Vigorous massage is injurious.

The rest-cure, as described by Dr. Weir Mitchell, is indicated for a certain class of neurasthenic women with decided impairment of nutrition and a complicating hysteria.

The discovery and treatment of the reflex irritations are important. After the stomach and liver, the pelvic organs and the eyes are the most fruitful sources of trouble.

The Sexual Neuroses and Psychoses.

Of the above disorders the neurologist has to deal chiefly with the vicious habit of masturbation (which may, however, be also a manifestation of disease) and the sexual neuroses, spermatorrhœa and impotence.

Masturbation and Spermatorrhœa.—Masturbation is the name given to the vicious habit of artificially exciting the sexual orgasm. It is very common among boys and less common but present among girls and adult men and women. It is usually only a vice due originally to low associations and teachings among children. In some cases it is a disease or the symptom of a neurotic or insane constitution.

Etiology.—It is most common between the ages of fourteen and eighteen, but may begin earlier. Even infants and very young children sometimes masturbate, usually as the result of some local irritation which leads them to rub the genitals. A tight prepuce, eczema, or worms may lead to the habit, but it is usually taught by a companion. The practice sometimes attacks schools almost like an epidemic, for in every institution a certain per cent of the boys are sexually precocious or vicious, while the others are ignorant and innocent of the evils of the practice. Masturbation is relatively rare after twenty, but is practised by some throughout life even up to old age.

Results.—Masturbation as ordinarily practised leads after a time to a feeling of malaise, mental depression, disinclination to work, study, or to enjoy one's self as before. The appetite is a little impaired, the extremities easily get cold and perspire readily. Peculiar numb feelings are felt in the hands and feet. There is an unnatural nervousness and irritability, and the power of concentrating the mind is a little weakened. The patients often have dilated pupils and hyperæsthetic skin. After a time nocturnal emissions occur. The organs become irritable and slight excitement causes erections. These symptoms may be slightly marked and pass away in a day or two, or until another indulgence occurs.

Masturbation is sometimes done to an extraordinary extent, even daily or twice daily for a considerable time. After a while the young man begins to find that he is not well and realizes that this habit is hurting him. Then if he be sensible and of healthy constitution he stops. Others are frightened out of it by friends or by reading the terrorizing stories printed in quack advertisements and circulars. Sometimes the fright thus caused leads the unhappy youth into a condition of hypochondriasis, which is helped on by the occurrence of nocturnal pollutions and the nervous debility resulting from his past indiscretions. In other cases in which there is a decided neurotic history, a genuine neurasthenia of a sexual type develops and annoys the patient for years.

Masturbation rarely leads to insanity and is oftener a symptom than a cause of such disorder. It is occasionally the cause of epilepsy. When this is the case the convulsive attacks are

likely to put on a hysteroid phase and are accompanied with peculiar co-ordinated convulsions and emotional disturbance. Masturbation is the common cause of hystero-epilepsy in women.

Diagnosis.—Many victims of the masturbation habit who have come to recognize its evils and tried to stop it develop a hypochondriacal condition, and feel sure that there is something in their faces which reveals to the world their trouble. This is not the case. But there is a certain physiognomy which in a measure characterizes the masturbator to such an extent that an experienced observer can detect it. The pale, pasty complexion, moist, furtive eye, dilated pupil, listless, restless, and depressed manner, the wet, flabby palms and hyperæsthetic skin, all help to tell the story. Locally the penis is often reddened and more or less turgid, the scrotum relaxed, and a varicocele may be present. Examination of the urine may reveal spermatozoa. The urine also is almost always of rather low specific gravity, and contains a great excess of phosphates, both earthy and alkaline.

Treatment.—The patient must be told plainly the necessity of stopping the practice. He must be impressed, but not terrorized. He should be kept out of doors at vigorous physical exercise, for sedentary and solitary work is always bad for such cases. He should be made to take cold-water baths and should sleep on a hard bed with light covering. He had better sleep with some one whose presence may exercise a controlling influence. He should not eat heartily at night, never just before going to bed, and what is still more important, he should not drink before going to bed. Sometimes it is well to have him wakened at an early hour in the morning, when he should empty his bladder; for emissions occur often early in the morning and are promoted by the irritation of a full bladder.

Locally cold-steel sounds may be introduced and allowed to remain for ten minutes three or more times a week, or the psychophor or Ultzman's short catheter may be used. In bad cases with a great deal of prostatic irritation, local applications of nitrate of silver are needed. Internally a mixture of tinct. opii, tinct. camph., and tinct. lupulin may be given at night, the ingredients being somewhat varied in amount to suit the case. Bromides, chloral, atropia, and salix nigra are also drugs which are often useful. The mechanical measures which have been devised for preventing erections, such as rings with sharp teeth, are rarely needed and rarely useful. They may even do harm by directing the mind to the affected function.

I do not believe it right for the physician to prescribe fornication. It is not safe nor curative, apart from the moral aspect of the matter. It has always struck me also as pretty small busi-

ness for a man to purposely select a wife to relieve him of the results of a weak will and vicious sensual indulgence. If marriage comes in the natural course of events, as it often does, so much the better. But to select a wife as a remedial agent for masturbation is unjust to the woman and a confession of moral and mental feebleness. Man is distinguished from the brute by his self-control. Let him bear this fact in mind and raise himself above the animals by a determined effort of the will. Pure thoughts and chaste associations, vigorous physical exercise and a resolute effort to act a manly part will always be successful.

Traumatic Nervous Affections (Traumatic Neuroses and Psychoses), Spinal Concussion.

The present tendency of neurology is to deny the existence of any special nervous affection produced by trauma or shock. There may follow from these causes:

1. Surgical injuries.
2. Neurasthenic states.
3. Hysterical states.
4. Hemorrhagic, inflammatory, and degenerative diseases.
5. Combinations of the foregoing.

These troubles may follow not only railway but other injuries, but are especially liable to follow those associated with intense fright.

2. Traumatic neurasthenia, or "traumatic neurosis," does not differ from forms of neurasthenia produced by other causes, except that with it there may be certain sprains and surgical troubles. Traumatic neurasthenia not infrequently is associated with much spinal pain, and in women especially the clinical symptom group known as "spinal irritation" may develop. In other cases there may be more of the features of spinal asthenia. When to these spinal symptoms a partial paraplegia is added, we have the disease that has been called "railway spine," or spinal concussion. The anatomical names given to this used to be spinal anæmia, spinal hyperæmia, and spinal meningitis. But no evidence has as yet proved the exact nature of the lesion.

3. Traumatic hysteria is a rare affection in this country. It does not differ from hysteria produced by other causes, except for its sudden onset and occasional surgical complications. It is usually a hysteria major and has the characteristic stigmata of that type. In this city electrical injuries and frights have produced some classical cases of hysteria major.

4. There is considerable evidence that in some rare cases traumatism may produce minute multiple hemorrhages throughout

the nervous centres. In such cases there are usually neurasthenic or hysterical symptoms and in addition symptoms of organic disease.

In the majority of cases the symptom complex is something like this (Knapp): "The patient has headache and vertigo; he is depressed, irritable, and hypochondriacal, with a diminished power of application; he may have some visual disturbance, he often has a contracted field of vision and occasionally optic atrophy; there is some tremor and perhaps inco-ordination; he has anæsthesia, usually not limited to one-half of the body, and with it numbness and prickling; his movements are slow and weak; his tendon reflexes are exaggerated; there is often some lack of control over his bladder; and he may have pain and stiffness in the back from muscular strain." The symptoms eventually resemble a disseminated sclerosis.

Massive hemorrhages and serious mechanical injury of the nervous centres may be also produced by injury.

Finally, it is a well-known fact that traumatisms may excite in the predisposed locomotor ataxia, inebriety, insanity, or may lead to the development of a cerebral tumor.

It is the mental impression, the shock, much more than the physical injury, which produces the neurosis or psychosis.

The symptoms may appear soon after the accident, or after a period of relative health lasting a few weeks the neurosis gradually develops.

The most important practical point in connection with the subject is the diagnosis and the elimination of malingering. This is additionally difficult for the reason that the hopes and anxieties depending upon litigation tend to cause introspection, exaggeration of symptoms, and unconscious bias even in the most honest. The opinion among American neurologists tends to favor the seriousness of traumatic neuroses. While malingering is not rare, yet if the patient has really a traumatic neurasthenia or hysteria the disease may not be a trifling one. Careful research, however, often tends to elicit the fact that previous to the injury the patient was an alcoholic, syphilitic, or neurotic, and perhaps had already the beginnings of his alleged traumatic disorder. In no part of clinical medicine is a careful and searching examination and weighing of symptoms more urgently called for. The methods of carrying out such examinations are given elsewhere. Special methods for testing anæsthesia are sometimes needed. The two sides of the body should be tested simultaneously with concealed needles, beginning on the trunk, or the faradic current with a double-pointed electrode may be used. There are few patients who can successfully deceive in an examination covering all the special senses.

The treatment of the traumatic neuroses calls for no special notice.

EXOPHTHALMIC GOITRE (BASEDOW'S DISEASE, GRAVES' DISEASE).

Exophthalmic goitre is a chronic neurasthenic neurosis characterized by rapid heart beat, enlargement of the thyroid gland, protrusion of the eyeballs, and various neurasthenic and vasomotor symptoms.

Etiology.—The disease occurs much oftener in women than men (four to one). It is a disease of early adult life, occurring chiefly between fifteen and thirty-five, very rarely in childhood, and never after fifty.* It is apparently more common in the Anglo-Saxon race, but is not very frequent in America. There is very rarely any direct inheritance of the disease, but the family is often a neuropathic one. As a rule, the patient is of a neurotic temperament. Anæmia and debilitating diseases promote its development. Goitre and heart disease do not seem to predispose to it. The most frequent exciting causes are powerful depressing emotions and severe physical exertion. Rarer causes are injuries and infectious diseases, such as measles, scarlet fever, and pneumonia.

Symptoms.—The disease usually begins gradually and the first symptom is in most cases rapid heart beat and palpitations, accompanied with some nervousness and tremor. The next symptom is enlargement of the thyroid gland, and at about the same time the eyeballs begin to protrude. This order of development does not always take place, and occasionally one of the three principal symptoms is not present. The disease is usually one or two years in developing, the heart symptoms being those which continue by themselves longest. With the symptoms mentioned there occur many minor troubles which are more or less characteristic. The patient is usually very nervous and irritable; a distressing insomnia may be present. There is almost uniformly a fine tremor (eight to nine per second) of the hands, less marked in the lower limbs and not present in the face or tongue. The reflexes are exaggerated. There is a tendency at times in walking for the knees suddenly to give way. The patient rarely has neuralgias, but does have burning or feverish sensations and headaches. The skin is rather reddened and the patient sweats profusely. Pigmentation and vitiligo are sometimes seen, and urticaria may develop. The electrical re-

*Among 33 cases at N. Y. Post Graduate Clinic (Fiske-Bryson) there were 8 males, 25 females. Ages: thirteen to twenty, 8; twenty-one to thirty, 8; thirty-one to forty, 6; forty-one to fifty, 5; fifty-one to sixty, 1.

sistance of the body is much diminished, being 800 to 1,500 ohms instead of 2,000 to 3,000. There is sometimes a dermographic skin, as in other neurasthenic states. The respiratory function is weakened and chest expansion often falls below one inch (Fiske-Bryson). Attacks of a persistent watery diarrhœa occur. Anæmia is usually present. There is occasionally polyuria, more rarely glycosuria. The menses are irregular and amenorrhœa often exists. A slight rise in temperature may occur.

The *major symptoms* of the disease are:

Tachycardia and pulsating arteries.
Goitre.
Exophthalmus.
Tremor.

The *minor symptoms* are:

Nervousness.
Sweating.
Insomnia.
Lessened electrical resistance.
Diminished respiratory expansion.
Subjective sensations of heat.
Diarrhœa.
Polyuria.

Symptoms in Detail.—Tachycardia is the most constant single symptom. The pulse beats from 100 to 120 per minute usually, but may rise to 160 or even 200. Its rhythm is usually steady; but palpitations occur easily, even without exciting cause. The patient may wake up at night with distressing attacks, something like those of angina pectoris, but the intense pain and sense of impending death are usually absent. The heart is dilated and a systolic murmur is often heard at the base propagated along the arteries. Real organic disease, however, is rare. The arteries are dilated and soft. They pulsate strongly, particularly the carotids. A thrill is sometimes felt over the heart and always over the goitre. The arterial tension is normal or low.

The thyroid gland is usually enlarged symmetrically; later in the disease the isthmus is affected and the three lobes of the gland stand out prominently (Fig. 198). If only part of the gland is involved it is oftenest the right lobe. A thrill is felt over it and a systolic murmur can be heard.

The bulging of the eyes or exophthalmus is usually bilateral and even. If one eye is alone or more affected it is the right. The exophthalmus varies much in degree. It is not usually very great, but may be so excessive as to prevent closing of the lids and to expose the insertions of the recti. The eyeball may be slightly enlarged (one-tenth). The pupils are normal and vision is not impaired, though myopia occasionally occurs. The fundus

and visual field are normal. Paralysis of some of the eye muscles is a rare complication. Weakness of the internal recti and exophoria are frequent. The lids show certain peculiarities. One of these, known as *Von Graefe's symptom*, consists in the inability of the lid to follow the downward movement of the eyeball. When the patient is told to follow the movement of the finger vertically downward the eyeball moves steadily, but the lid catches, as it were, and refuses to follow or does so in a jerky manner. Another symptom, known as *Stellwag's symptom*, is a considerable retraction of the lids, especially the upper one. Both this and Von Graefe's symptom are due to a common tendency

FIG. 198.—EXOPHTHALMIC GOITRE, WITH AND WITHOUT EXOPHTHALMUS.

of the lids to retract—due perhaps to overaction of the muscle of Müller. A tremor of the lids sometimes occurs.

Course.—The disease progresses slowly. After a year or two it often becomes stationary for a long time. Cases of gradual spontaneous recovery occur. The natural duration of most recoverable cases is two to four years. In those which do not recover the disease lasts five, ten, or more years. Eventually the patient emaciates, the heart becomes weaker, albuminuria and dropsy appear, diarrhœa sets in, and the patient dies of exhaustion or is carried off by phthisis or some intercurrent disease. Other cases having improved up to a certain point remain in this state for years.

Complications.—Mental derangement occasionally occurs in the later stages of the disease. Hysterical crises, epileptic at-

tacks, choreic movements, paralysis of the ocular muscles, muscular atrophy, paralysis agitans, Addison's disease, diabetes, locomotor ataxia, and local œdema have all been observed. With the exception of hysterical attacks these complications are rare.

Abortive Forms.—This name is applied to cases in which only a part of the distinctive symptoms develop. Tachycardia always exists; with it are tremor and moist skin, lessened electrical resistance and nervousness. Or tachycardia and goitre may alone be present.

Pathology.—The disease may be regarded as a neurasthenic or atonic condition of the vasomotor centres and of the great visceral nerves, the vagus and spinal accessory. As a result of the first condition the blood-vessel walls are relaxed and the cutaneous hyperæmia results. The enlargement of the thyroid is explained by a vasomotor paresis of its vessels. It is a kind of erection of the organ. The exophthalmus is also due chiefly to paralysis of the orbital vessels. A tonic spasm of the muscle of Müller is thought to help in producing this symptom. This muscle consists of unstriated fibres originating in the membranous lining of the orbit and inserted into the lids. It is rudimentary in man, and its influence in causing protrusion of the globe must be very small. The deposit of retrobulbar fat is a secondary phenomena. The rapid heart beat is probably due to impairment of the inhibitory fibres of the spinal accessory. Thus all the symptoms may be explained on the theory of a vasomotor and cardio-inhibitory paresis.

Another theory suggested of late is that the disease is primarily an affection of the thyroid gland; that this organ is overactive and throws into the blood substances that irritate the nerves. More properly the mechanism should be that it withdraws from the blood too much of those substances which are needed for nerve nutrition. The principal substance so far as known which develops in excess when the thyroid fails to act is mucin. In myxœdema the thyroid gland atrophies, and a long list of symptoms almost directly the opposite of those of exophthalmic goitre occurs. The thyroid theory is weakened by the fact that tachycardia and many other characteristic symptoms may develop without a goitre, and that in simple enlargement of the thyroid the symptoms of Graves' disease do not appear.

Small hemorrhages and congestion of the medulla have been found in a few cases. Aside from this, no important changes in the central nervous system have been discovered. The heart is dilated and enlarged, endocarditis is sometimes present, oftener not. The arteries are dilated. The thyroid gland is vascular and its arteries dilated; its proper structure is increased in amount. There is increase of fat and vascular dilatation in

orbit. The thymus gland is usually present to a greater extent than normal.

Prognosis.—About one-fifth of the cases get well or practically well. Probably over half the cases, if they can be properly treated, reach a fairly comfortable condition of improvement. The cases in which symptoms come on quickly have the most favorable prognosis. In those with marked exophthalmus and goitre the prognosis is not so good.

Diagnosis.—The disease can be distinguished by the persistent tachycardia, with goitre or exophthalmus, and in its early stage by the tachycardia with tremor, moist skin, sensations of heat, nervousness, insomnia, lessened respiratory expansion, and electrical resistance. A symptomatic Graves' disease may sometimes be caused by a goitre pressing on the vagus or sympathetic and causing irregular heart beat and perhaps exophthalmus. In these cases the history of a long-standing goitre exists, the heart's action is irregular, the exophthalmus is usually partial and one-sided. In abortive forms it is necessary to have tachycardia and at least one other of the four major symptoms to make a diagnosis.

Treatment.—Rest is the most important single thing. The patient should be put to bed or kept on the back for one or more months. Freedom from excitement and worry must be enjoined. No especial diet is needed, nor do climatic influences or baths or mineral waters have much effect. Some cases are said to be improved, however, by removal to heights of one to three thousand feet. In most cases a sea-voyage is the better change if one is made.

The drugs used are numerous. The most efficient are tincture of strophanthus in doses of fifteen to forty drops daily, iodide of potassium or the syrup of hydriodic acid, arsenic and bromide of potassium used together; quinine, mineral acids, and iron, used together. Other remedies are tincture of aconite in ♏ v. or aconitia gr. $\frac{1}{200}$ doses, tincture of belladonna increased to the limit of tolerance, tincture of cactus grandiflorus in ♏ x. to xx. and tincture of veratrum viride, ♏ x. to xxx. dose, and the picrate of ammonia, gr. i. to ij. t. i. d. Of these drugs, strophanthus, the iodides, bromides, and iron have served me best. Digitalis is of doubtful value.

Electricity possesses some utility. It should be given, if possible, two or three times daily in the form of galvanism and in doses of two to six milliamperes for ten minutes. The technique is as follows. Positive pole on back of neck, negative drawn along course of vagi in the neck; each side two minutes. Same with positive pole placed subaurally one minute; negative pole over thyroid two minutes, negative over cardiac region one min-

ute, positive pole over eyes, negative over thyroid one minute, two milliamperes. The faradic current may be used for general tonic effects or combined locally with the galvanic. The patient should lie down during treatment and remain quiet for an hour later.

For the palpitations, sulphate of sparteine or strophanthus with Hoffmann's anodyne may be used. The ice-bag placed over the heart and neck is helpful and may be used systematically. Tonic hydrotherapy is often useful, but should be carefully employed. Surgical treatment until late years has been unsuccessful. Recently several cases have been reported in which cure has been produced by partial removal of the thyroid gland. This measure is yet in an experimental stage. Treatment of the nose has been said to cause disappearance of symptoms, but its utility is very doubtful. Respiratory exercises by which the patient is taught to increase his chest expansion do much good, as shown by Dr. Bryson.

Mild compression of the lids at night seems to help the exophthalmus, and slight and steady compression of the thyroid gland sometimes reduces its size a little.

CHAPTER XXV.

PROFESSIONAL NEUROSES, OCCUPATION NEUROSES (WRITERS' CRAMP AND ALLIED AFFECTIONS.)

Writers' cramp is a chronic functional neurosis characterized by spasmodic, tremulous, inco-ordinate or paralytic disturbance when the act of writing is attempted, and associated with feelings of fatigue and pain.

Etiology.—It is a disease of the present century, and has been particularly noted since the introduction of steel pens about the year 1820. A neuropathic constitution is often present, and sometimes there is a hereditary history. Men are much more subject to the disease than women. The most susceptible age is between twenty-five and forty. It rarely occurs after fifty or before twenty. Clerks and professional writers are naturally much more subject to the disease. Excessive worry, intemperance, and all debilitating influences predispose to it. The chief exciting cause is excessive writing. But this is not all. The writing that is done under strain or a desire to finish a set task is the harmful thing. The style of writing is also an important factor. Writing done in a cramped posture with movements of the finger alone or with the little finger or wrist resting on the table is most injurious. Free-hand writing done from the shoulder according to the American system is least harmful. Shaded or heavy writing with sharp steel pens is also productive of harm. Copying is much more harmful than composing. Authors seldom have writers' cramp. Albuminuria, lead-poisoning, exposure to wet and cold, and local injuries are sometimes exciting causes.

Symptoms.—Writers' cramp very rarely attacks a person suddenly. The patient first notices a certain amount of stiffness occurring at times in the fingers, or the pen is carried with some uncertainty and jerky movements are made. He feels a sensation of fatigue in the hand and arm, and this may amount to an actual tired pain. The first symptoms may last for months or even years. The hand is rested as much as possible, new pens or penholders and new modes of holding it are tried. Often the patient, fearing the onset of the cramp, and as its result loss of employment, becomes anxious, worried, and mentally depressed.

Sometimes the trouble is worse when beginning a daily task, and it gradually wears off in a few hours. At other times exactly the reverse is the case. When the disease has reached its highest stage, writing becomes almost or entirely impossible. The moment the pen is taken in the hand and an attempt at using it made, spasmodic contractions of some of the fingers, or even of the arm, occur, the pen flies in any direction, and it is impossible to control or co-ordinate the movements. The rule is that although writing cannot be done, all other complex movements are performed as well as ever. Thus the sufferer from writers' cramp may be able to play the piano, or paint, or thread a needle, or use the hand in any complex movements. This limitation, however, is not always present. Telegraphers, who use to some extent the same muscles as in writing, and who also often have to do a great deal of writing, are liable to suffer from both writers' and telegraphers' cramp at the same time. No evidences of actual paralysis are present in the affected muscles, and there is rarely anæsthesia, but the arm aches and is sometimes tender. Sensations of numbness and prickling are present: in rare cases vasomotor disturbances are observed; associated muscular movements of the other arm or of the neck or face sometimes occur. The hand may tremble on attempting to write or fall almost paralyzed when the pen is taken.

The various symptoms occur with different degrees of prominence, so that the disease has been classed under the heads of (1) the spastic, (2) the neuralgic or sensory, (3) the tremulous, and (4) the paralytic forms. These forms are, however, often more or less mixed.

1. The spastic form is undoubtedly the most common, and it has given to the disease its name. Cramp of some muscle or muscles is present in over half of the cases. The muscles of the thumb and first three fingers are oftenest affected, and in some cases the flexors, in some the extensors, are chiefly involved. In telegraphers' cramp it is the extensors, but in writers' cramp the flexors, that are mainly attacked. The thumb or forefinger or the little finger alone may suffer from the spasms. The pronators and supinators are quite often involved. The spasm is usually a tonic one. With the spasm there is also inco-ordination so far as writing movements are concerned, and this fact is quite as important in producing the bad writing as the spasm. The inco-ordination is apparently of the motor type, and is not due to anæsthesia of the muscle sense, as in locomotor ataxia.

2. The neuralgic form resembles the spastic plus sensations of fatigue and pain, which are quite severe and are brought on by writing. There may be tenderness along the arm also.

3. The tremulous type, though rare, is very characteristic

when present. The patient when attempting to write observes a tremulous movement of his hand and arm. This ceases when his attempts to write cease. The tremor usually affects most the fingers used in pen-prehension, but it also spreads to the forearm and may even involve the entire extremity. An oscillatory or lateral tremor, due to involvement of the pronators and supinators, has been observed. The tremor is of the character known as "intention-tremor," such as is observed in disseminated sclerosis. It is shorter in range and more rapid than the tremor of that disease.

4. The paralytic form, or that type in which muscular feebleness is the dominant symptom, is said to be rare by Gowers, and this accords with my experience. German writers speak of it as common. In the typical paralytic form the patient, as soon as he begins to write, feels an overpowering sense of weakness and fatigue in the fingers and arm. The fingers themselves loosen their grip and the pen may drop from the hand. Powerful impulses of the will and change in the mode of holding the pen enable the sufferer to continue, but the arm aches and finally is absolutely painful, and weakness and fatigue compel the writer to desist. Sometimes the paretic condition is succeeded by the spastic. Many of the cases of paralytic writers' cramp are not true examples of the neurosis, but are rather cases of neuritis of a rheumatic or other type.

General Symptoms.—Writers' cramp is essentially a motor neurosis, and its leading symptom is the impairment of a motor function. Other symptoms, however, both general and local, are always associated with it. These are mainly (1) psychical and (2) sensory, more rarely (3) vasomotor and (4) trophic.

1. Psychical symptoms. The patient is often nervous, emotional, and mentally depressed at times. He suffers from insomnia and vertigo. Patients are generally unwilling to admit that there is any other trouble than the local one, and only careful examination may bring evidence of constitutional trouble. There are cases of purely mental writers' cramp.

2. Sensory troubles. These consist of pain, sense of fatigue, feelings of numbness, prickling, pressure, weight, tension, constriction, etc. Hyperæsthesia, and more rarely anæsthesia, are also observed. The most common sensory symptom is that of aching and fatigue, and this is usually confined to the arm, and oftenest runs along the course of the radial and median nerves. The cervical vertebræ may be tender, and sometimes patients have a headache in the parietal region of the side opposite the affected arm.

3. Vasomotor, trophic, and secretory disturbances. The condition known as *digiti mortui* has been observed, coming on par-

oxysmally. It is a symptom which the general neurasthenic state helps to produce. When the nerves are involved decided vascular changes may occur, such as passive congestion of the hand and arm, with swelling and turgescence of the fingers, and a sensation of throbbing. In bad cases the fingers will look as if they had chilblains. Local sweating, dryness of the skin, and cracking of the nails, all are conditions which may follow impairment of writing power from neuritic causes.

Electrical Reactions.—The results of observations upon the electrical reactions of the affected parts are somewhat contradictory. Ordinary tests will, as a rule, reveal very little change. Sometimes there is a quantitative increase, sometimes a decrease, of irritability to both forms of current. The increase occurs in the earlier stages, the decrease in the later. An increase or modification of electro-muscular sensibility has been noted. The electrical examinations, therefore, are only of value in excluding a neuritis or possibly in determining the stage of the disease.

Pathology.—Neuritis is undoubtedly present in some forms of writers' cramp, so called. It is not present, however, so far as external tests go, in the typical neurosis. Nor are there any post-mortem observations throwing light on the anatomy of the disease. We must believe, therefore, that it is a neurosis having no appreciable anatomical basis.

The act of writing is a very complicated one, calling into play numerous sets of delicately innervated muscles. These muscles are employed: 1, in pen-prehension; 2, in pen movement; 3, in holding the arm and wrist tense.

1. The muscles employed in pen-prehension are the two outer lumbricales, two outer interossei, the adductor muscles of the thumb, the flexor longus pollicis; to some extent the deep and superficial, short and long flexors, and the extensors of the thumb. These are supplied mostly by the ulnar (interossei, adductor pollicis, inner heads of deep flexor of fingers, and inner head of short flexor of thumb). The rest of the muscles are supplied by the median.

2. In moving the pen, if the writing is done mainly by finger and not by arm movements, the muscles brought into play are the flexor longus pollicis, extensor secundi internodii pollicis, flexor profundus digitorum, extensor communis digitorum, and to some extent the interossei. The musculo-spiral and ulnar nerves innervate these groups about equally. In moving the pen by the "American" or free-hand method there is a very slight play of the above muscles, while most of the pen movement is done by the muscles of the upper arm and shoulder, viz., the teres major, pectorales, latissimus dorsi, biceps, and triceps.

The spinal centres for these muscles are distributed along the fifth, sixth, and seventh cervical segments of the cord. The cells are larger and situated more superficially in the anterior gray horns.

3. Besides these movements involved in pen-prehension and in the letter-making, a certain amount of muscular tension is exercised in "poising" the forearm and hand and steadying the wrist. The biceps and triceps, the supinators and the flexors, and extensors of the hand are here brought into play.

From the foregoing it will be seen that the muscles of pen-prehension are most used in all but the free-hand style of writing, since the same groups have a double duty, that of clasping and of moving the instrument.

While writers' cramp is often complicated with some neurotic disturbance leading to associated symptoms of pain, paralysis, tenderness over nerves, vasomotor disturbances, etc., there can be no doubt that the lesion in typical cases is central. Writing is an acquired automatic movement, and it must have as its anatomical basis a certain established arrangement of nerve-cell groups in the cervical part of the spinal cord. The nerve impulses generated in the cerebral cortex pass along the pyramidal tracts and set at work those ganglion groups which, in turn, innervate the muscles used in writing with motor impulses. These groups are themselves innervated also sensorially by the nerves of muscle sense in movement. In writers' cramp the spinal groups of cells are more or less used up or exhausted, and the motor impulse which naturally would innervate them strikes cells which respond unequally, or it overflows to other cell groups, and hence the spasmodic, irregular movements of the arm. The cerebral centres in the cortex or basal ganglia are closely connected with those in the cord, and may be said to form part of the writing mechanism. It happens in some cases that this centre too is exhausted and discharges its impulses irregularly. In some cases, therefore, the cerebral, and in some the spinal, cell groups are the more affected, and a diagnosis of the exact condition in each is approximately possible and may be not without practical importance. Writers' cramp is a disorder of efferent paths and stations. The muscular sense does not seem to be involved.

The pathology of writers' cramp is that of all the other forms of occupation neuroses, and nothing need be said upon this point regarding them when they come to be considered.

The *diagnosis* of well-marked cases of writers' cramp presents no difficulty. In the earlier stages, however, it may be confounded with a large number of disorders, viz., post-hemiplegic chorea, hemiataxia, progressive muscular atrophy, progressive locomotor

ataxia, various forms of tremor, lead paralysis, rheumatoid arthritis, neuritis, cerebral and nerve tumors, and tenosynovitis.

In many of these cases it is only necessary to bear in mind the history of the disease in order at once to reach a safe conclusion as to its nature.

If there is a great deal of pain in the arm, with tenderness along the course of the nerves; if there is decided change in the electrical reactions; if there are sensations of tingling, numbness, etc.; and if the patient shows an absolute loss of power in the various groups of muscles, with some incapacity for doing other acts besides the one with which he is specially concerned, then the trouble is undoubtedly peripheral and due largely to an underlying neuritis. The prognosis in these cases is much more favorable. If, on the other hand, the disorder comes on in persons who have done an excessive amount of writing; if it is associated with nerve strain; if the electrical reactions are but slightly changed, the sensory symptoms slight, and the motor inco-ordination marked, limited to the special class of work, and not accompanied with absolute paresis, the disorder is central and needs both a different treatment and prognosis. It is these cases that form writers' cramp proper, although no doubt neuritic and central forms are associated, or the former may run into the latter.

Course and Duration.—Writers' cramp is a chronic disease. It begins insidiously and attacks one group of muscles after another as each is brought into play by new methods of writing. If the left hand is used, that, too, is liable to become affected. The course varies, however; for a time progress may be arrested or improvement set in. When the disease becomes well established it will most often last a lifetime.

Prognosis.—The prognosis is unfavorable, yet not so much so as has once been thought. Undoubted cases of complete recovery have been reported, even under unfavorable conditions.

The prognosis is much more favorable if the patient begins treatment early and before marked spastic symptoms are present. It is more favorable in the neuralgic forms. Some patients who suffer from a mild form of the trouble manage, by the help of instruments or special pens, to do their work for years. The more acute the disease and the more evidently peripheral and neuritic its origin, the better the prognosis. In over one-fourth of the cases, patients who use their sound arm will not be affected in it.

The facts stated regarding the cause, physiology, and general symptomatology of writers' cramp apply to the other forms of occupation neuroses. A few special details, however, will be given regarding these. The most common and important are musicians' cramp and telegraphers' cramp.

Musicians' Cramp.—Under this head we include pianists' cramp, violinists' cramp, flutists' cramp, and the cramp of clarionet players.

Pianists' cramp occurs usually in young women who are studying to become professionals or who are especially hard-working and ambitious. The absurd "Stuttgart method" of teaching the piano, in which the motions are confined as much as possible to the fingers, predisposes especially to this disease. The symptoms are those of fatigue, pain, and weakness. The pains are of an aching character. They are felt in the forearm especially, but extend up the arm and between the shoulders. Spasmodic symptoms are rare. The right hand is oftener affected, but both hands eventually become involved.

Violinists' cramp may attack the right hand which holds the bow or the left hand which fingers the strings, but more often the left hand is affected.

Clarionet players sometimes suffer from cramp of the tongue and of the laryngeal muscles.

Flute players suffer not very infrequently from slight laryngeal spasms. A similar trouble affects elocutionists. The term mogophonia is applied to this type.

Telegraphers' cramp affects especially those operators using the Morse system, an instrument which is still the one most widely in vogue. Contrary to the opinions of previous writers, Dr. Lewis believes that this neurosis is not a rare one and is destined to become more frequent. In this city the cramp is not rare, the proportion being about one in every two hundred. The technical name among operators for the cramp is "loss of the grip." In telegraphing, the extensors of the wrist and fingers are called most into play, and hence are most and earliest affected. The symptoms come on very slowly, the thumb and index-finger being first affected. The victim finds that he cannot depress the key on account of spasm in these muscles, and he finds most difficulty in making the dot characters, such as h (. . . .), or p (. . . . .), or z (. . . .). When the flexors are most affected the key is depressed with undue force and a dash is made instead of a dot. Sufferers from the "loss of grip" generally suffer from writers' cramp also. While spasm is usually present, the disease may show itself simply in pain, paresis, and incapacity to co-ordinate the muscles.

In *sewing spasm*, which affects tailors, seamstresses, and shoemakers, clonic and tonic spasms attack the muscles of the hands on attempting to use them in the regular work. Tailors who sit cross-legged sometimes suffer from a peculiar spasm on assuming this position. It is possible, however, that these are cases of tetany, and not the functional neurosis under consideration.

Smiths' spasm, hephastic hemiplegia, appears to have been observed only by Duchenne and Dr. Frank Smith. It occurs in persons engaged in pen-blade manufacturing, saw-straightening, razor-blade striking, scissors-making, file-forging, etc. In doing this work they have to use a light or heavy hammer, with which strokes are delivered very rapidly and carefully. After a time spasmodic movements occur in the arm used, and the arm falls powerless. As in the cases reported there are generally hemiplegic symptoms, and also neuralgias, vertigo, and other cerebral troubles, the disease cannot be a pure "occupation" neurosis.

Drivers' spasm has been observed in veterinary surgeons by Dr. Samuel Wilkes.

Milkers' spasm is an extremely rare affection, which was first described by Basedow and seems to occur in milkmaids, never in milkmen.

Cigarmakers' cramp is very rare.

Watchmakers' cramp and *photographers' cramp* are also to be regarded merely as pathological curiosities.

Ballet-Dancers' Cramp.—Under this name certain painful and paralytic troubles occurring in ballet-dancers, especially premières danseuses, have been described by Schultz, Onimus, and Kraussold. It does not appear that the trouble is really a co-ordinative functional one, but is rather neuralgic, or the result of local strain upon the parts.

The list of professional neuroses is made to include, besides those above given, cramps and co-ordinative troubles affecting artificial-flower makers, billiard-players, dentists, hide-dressers, electrical-instrument makers, stampers, turners, sewing-machine girls, money-counters, weavers, painters, and pedestrians.

Prophylaxis and Treatment.—The introduction of typewriters, gold pens, and improved penholders has prevented somewhat the increase of writers' cramp. Stenographers rarely have it unless they write in long hand. Persons who have to write a great deal should use large cork or rubber penholders and gold or quill pens with smooth paper. The best style of writing is that done from the shoulder, but this is a method that book-keepers and those who have to keep accounts cannot easily adopt. Many nervous persons have a bad habit of gripping the pen very tightly and pressing down on the paper with excessive force. Fatigue soon results and painful sensations develop in the arm. Proper attention should be paid to the position of the paper written upon, the height of the desk, the light, and the sleeves of the coat or dress. The paper should be laid at an oblique angle to the edge of the desk, and not at a right angle as many writing-teachers are accustomed to direct. As some cases of "cramp" are undoubtedly cerebral, it is very unwise to attempt any extraordinary exploits in writing or to work with the ambition to put the writing capacity to the utmost test. Cramp is often dated from days when such extra work is done.

When the cramp is fully developed, the most essential thing is rest. This may be secured by getting a new form of penholder, holding the pen in a different way, using the unaffected arm, using some form of mechanical appliance. The mechanical appliances are splints, rubber bands around the wrist, and various instruments contrived to prevent spasm and throw the work of writing on new and larger groups of muscles.

Instruments for writers' cramp are very numerous. Those that are of some value are Mathieu's, Nussbaum's, and some modification of Cazenave's (see Figs. 199, 200). All the various instruments have been of service, or have even been curative in some

special cases, but not too much must be expected of them. As a rule they are only palliative. A cheap instrument that may prove satisfactory is that of Mathieu.

In the medical treatment of writers' cramp, the two most important agents are massage and electricity.

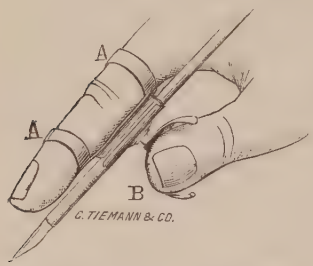

Fig. 199.—Mathieu's Instrument for Writers' Cramp.

By massage only very mediocre results were obtained until greater attention was drawn to it by Mr. J. Wolff, a writing-master of Frankfort-on-the-Main. This gentleman has cured many cases, though not all that he has treated (Berger), and he has secured many testimonials for his method. The treatment, as described by Schott (G. W. Jacoby), consists of a system of gymnastics and masage. The gymnastics consist of movements performed by the patient alone and movements executed with the co-operation of the operator. The first are performed by the patient during from twenty to thirty minutes, rarely for forty-five minutes. These movements consist of gymnastics of the fingers, extension, flexion, abduction, and adduction being performed, and the thumb being exercised separately. After this the same four motions are executed at the wrist joint, then extension and flexion of the forearm, and ultimately the arms themselves are exercised in the same manner and are to be lifted over the head. Each single exercise is to be performed from six to twelve times. After each motion a pause is to be observed. The opposed movements are to be carried out in the same manner, except that the operator must carefully resist their execution as though he were endeavoring to force the patient to perform a motion just the reverse of his intentions. Regularity of pressure is to be observed in this, so that the same amount of force is always used and so that the pressure does not vary in intensity from moment to moment. The time to be devoted to these opposed movements should be the same as that for the unopposed ones. According to the intensity of the affec-

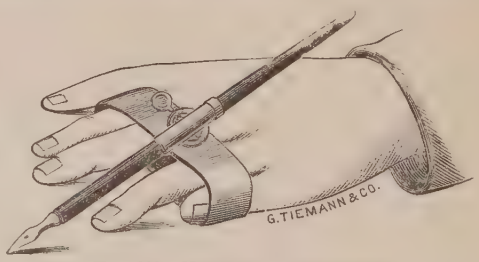

Fig. 200.—Nussbaum's Instrument.

tion, the exercises must be repeated two to three times daily. The massage itself consists of two parts—nerve and muscle massage. The nerve massage is effleurage along the course of the nerve trunks, the median, ulnar, and radial, going upward to the axillary and cervical plexuses. This effleurage lasts about ten minutes. Following this is the muscle massage. This consists of pétrissage, beginning with the hand and ending at the shoulder. The duration is the same as that of the last movement. One sitting a day has always proved sufficient. Wolff, in addition, uses "a peculiar method of writing instruction" and employs rubber bands and rings in his manipulations.

Electricity ranks second to massage in the treatment of occupation neuroses. The faradic current may be used when the affection is paralytic, but the galvanic current is usually more efficacious. It has been almost uniformly helpful in my experience. It must be given daily, however, or even twice a day. The anode is placed over the cervical spine and the cathode over the various muscular groups affected. A stable current of five to ten milliamperes for from ten to fifteen minutes is given. Galvanization of the sympathetic is recommended by some. Galvanic belts applied about the arm seem irrational enough, but have been known apparently to effect cures.

Lotions containing muriate of ammonia, liniments, hot and cold douches, the cautery, all have been recommended in professional neuroses. Tenotomy was once employed, but has been abandoned. Very little can be expected of drugs. The most trustworthy are atropine, strychnine, cannabis indica, the iodides and bromides, and cod-liver oil. It should be remembered that sometimes the disease is almost purely cerebral, and then an anti-neurasthenic treatment is called for. But in other cases, when the disorder is largely peripheral, the usual treatment for a low grade of myoneuritis must be employed.

CHAPTER XXVI.

PARALYSIS AGITANS (SHAKING PALSY, PARKINSON'S DISEASE).

Paralysis agitans is a chronic progressive disease, characterized by tremor, muscular rigidity and weakness, and by a peculiar attitude and gait together with sensations of heat, pain, and restlessness.

Etiology.—It occurs oftenest between the ages of fifty and sixty, then between sixty and seventy and forty and fifty. In very rare instances it occurs in early life, but the genuine disease does not occur before puberty. Males are affected much oftener than females (five to three in seventy-eight American cases). It occurs in all clases of life, but oftener among those who incur exposures and endure hard labor. It is not a disease of vice and is not the result of alcoholism, syphilis, or sexual excess. Heredity is a rare factor, but I have known hereditary family tremor to end in paralysis agitans. It appears to have some relation to rheumatism and especially to rheumatoid arthritis. It occurs oftenest in this city among the Irish, German, and Polish races (twenty Irish, thirteen Germans and Russians, the last mostly Hebrews).

The exciting causes in the majority of cases are exposure to wet and cold, fright, injury, and prolonged mental anxiety. An attack of sciatica or rheumatism, a sudden severe muscular strain, and fevers are rare causes. I have never been able to trace a case to malaria.

Symptoms.—The disease usually begins slowly with some aching pains in the arm and a slight tremor in the fingers of one hand, oftener the left. This gradually extends and involves the foot of the same side, then the other side becomes affected. The neck, face, and tongue are rarely attacked, and then to a small extent. After or with the tremor there comes on a stiffness in the arms and legs, and indeed of the whole body. With this there is a general contracturing and shortening of all the flexor groups; so that the head and body are bent forward, the fingers are straight but are flexed as a whole on the metacarpus, the forearms flexed on the arm, the trunk flexed forward on the thighs, and the knees slightly bent. The attitude gives the idea of extreme senility (Fig. 201). The gait is slow, the steps short and

shuffling; the patient has trouble in starting, stopping, and turning corners, owing to the slowness in initiating new movements in the voluntary muscles. When once started he may be unable to stop and has to run along. The speech early becomes affected. The voice is high-pitched, weak, and piping or senile in quality. There is a slowness in getting out words or in starting a sentence, though after it is begun the words come rapidly. The condition is analogous to the hesitation in the gait.

Along with the other symptoms there are often, though not always, sensations of heat, burning, fever, and rarely of coldness. These sensations are felt most in the feet, legs, or arms diffusely. Often there is a general feeling of restlessness and nervousness.

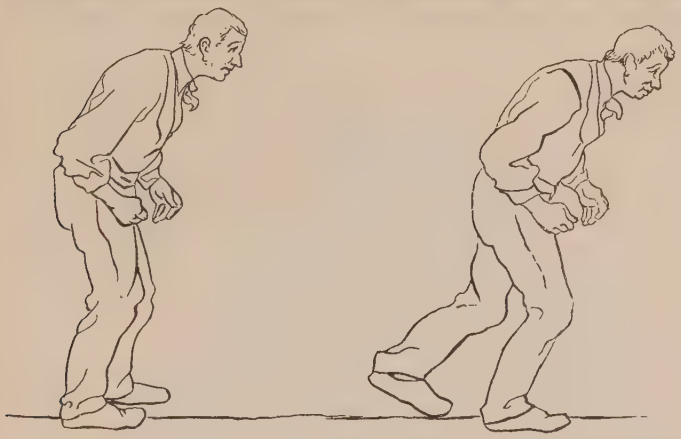

FIG. 201.—ATTITUDE AND GAIT IN PARALYSIS AGITANS.

Aching pains and a sense of fatigue occur; neuralgic pains are more rare. There is always a peculiar redness and flush in the faces of the patients. Sometimes they sweat profusely. Their temperature in the axilla is normal, on the skin it is sometimes increased (Peterson). The appetite is excellent, often abnormally great, and digestion is good. Visceral complications are rare. Muscular weakness comes on early; it slowly increases, but complete muscular paralysis does not occur. The disease ends in rigidity, which makes the patient as helpless as if paralyzed, but the muscles preserve considerable functional power to the last. The deep reflexes are present and not, as a rule, exaggerated; but exaggeration and even clonus occur in a small percentage of cases. As the disease progresses the tremor increases in extent, and continues without remission during all the waking hours; the limbs get more rigid; the patient becomes bedridden and is finally carried off by exhaustion or some intercurrent illness (Fig. 202).

It will be seen that the dominant symptoms in paralysis agitans are:

1. Tremor.
2. Rigidity, progressively increasing.
3. Sensory and vasomotor disturbances.

Further details must be given regarding these symptoms:

The *tremor* is at first rather fine, but later is coarse. It ranges from about 6 vibrations per second to 3.7. The average rapidity is 4 or 5 per second, which is about one-half the normal muscular rhythm. But the chief characteristic of the tremor is that it continues when the hand or limb is at rest, while voluntary motion causes it to cease. As the hand rests on the knee it shakes; as it is moved the tremor stops. When held straight out there is no shaking for a moment, but it soon begins. A glass of water is carried safely to the lips. The patient can control the tremor for a moment, especially in the early stages of the disease. These facts about the tremor apply in ninety per cent of cases. But there are patients whose tremor is slight when the limb is at rest and is increased on voluntary effort. The hands are affected in a characteristic way. The fingers and thumb are slightly flexed and held about in the writing position; the tremor moves the fingers and thumb as a whole, and they vibrate so that the one pats the other gently. Sometimes the tremor is one of alternate supination and pronation of the forearm. The neck and face muscles are not usually or extensively involved, the shaking of the head being generally the result of the general bodily tremor. Sometimes one sees a tremor of the lips or neck muscles. The tongue and eye muscles are practically never involved.

Rigidity.—The tremor may be almost or even entirely absent in this disease. If so, there is a corresponding dominance of the symptom of rigidity. The rigidity, as stated, comes on early, and may be the first and even the only prominent symptom. It affects chiefly the flexors of the arms, head and trunk, and legs, producing a characteristic senile position. In rare cases the extensors of the neck are affected and the head is drawn back. Cramps occur, and there is always a sense of stiffness. The muscular movements are slow, especially the initiation of a movement. Once started, a motion may be quickly done. The gait is peculiar: the steps are short and shuffling; the patient may have difficulty in starting, but once started he goes along well; or while walking there may be a sudden running forward. This is called "festination." Rarely there is a tendency to run backward or sideways. The facial muscles are stiffened and little used, so that the face has a peculiar expressionless look. The patient is often emotional, but the mind is not seriously affected. The

urine is usually about normal, but contains an excess of phosphates. There may be polyuria and less often glycosuria.

Course and Duration.—The disease slowly but steadily progresses until a full development of symptoms occurs, when it may remain stationary. It takes about two years for the whole body

FIG. 202.—TERMINAL STAGE OF PARALYSIS AGITANS, showing rigidity and contractures.

to be affected, though this varies much. It lasts from three to twelve years. In three cases of mine death occurred in three, six, and eleven years. It may last for thirty years or even more. Death is due to exhaustion and may be accompanied with mild delirium and fever.

The *diagnosis* must be made from senile tremor, multiple sclerosis, post-hemiplegic tremor, and wry-neck affecting the ex-

tensors bilaterally (retrocollic spasm). Senile tremor occurs in the very old and affects the head first and most. In multiple sclerosis the tremor is more jerky and is a tremor of motion; there is nystagmus, syllabic speech, and often apoplectiform attacks, eye trouble, and paralyses.

Post-hemiplegic tremor is accompanied with a history of hemiplegia; there are paralysis and exaggerated reflexes and the disease is unilateral. In retrocollic spasm only the neck muscles and frontalis are involved. The absence of exaggerated reflexes, the peculiar voice, gait and attitude, and the sensations of heat and nervousness often help greatly in the diagnosis.

The *prognosis* is favorable as regards life; unfavorable as

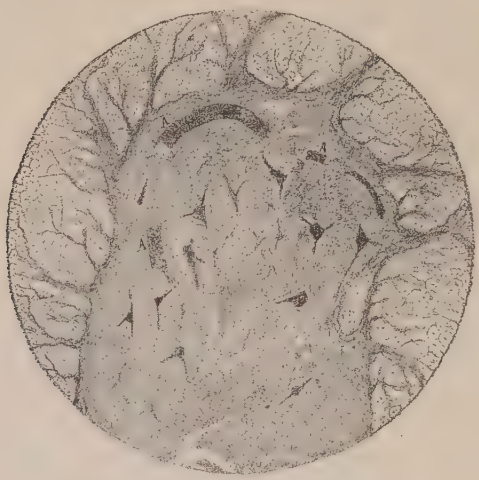

Fig. 203.—Anterior Horn of Spinal Cord, showing dilated capillaries.

regards cure; moderately good as to bringing about a cessation of progress in the symptoms.

Pathology.—The following are among the changes that have been found in the central nervous system: Hypertrophy of the nerve cells of the pons, induration of the pons, medulla, and cord, thickening of blood-vessel walls and of the pia mater, increase of connective tissue in the motor tracts, nerves, and muscles. In some cases autopsies have revealed nothing. The most constant change described is an increase of connective tissue in the motor tracts and pons, with congestion and hyperplasia of the vessel walls. I have found a peculiar dilatation of the vessels of the anterior cornua and roots, with some atrophy of motor cells (Figs. 203, 204).

Everything points, however, to the seat of the disease being primarily in the upper motor or cerebro-spinal segment.

Forms.—The unusual types of paralysis agitans are the hemiplegic or the monoplegic, the rigid type, and the retrocollic type. The only one of importance is the rigid type, in which there is practically no tremor.

Treatment.—The most important measure is rest, mental and physical, with plenty of fresh air. No special diet is indicated. Lukewarm baths and mild massage are agreeable and helpful. I know of no climatic cure. The galvanic current produces temporary relief; it should be given daily. Hyoscine hydrobromate, first used by Charcot and introduced into this country by Seguin, is of much temporary value in relieving the tremor (gr. $\frac{1}{100}$ in-

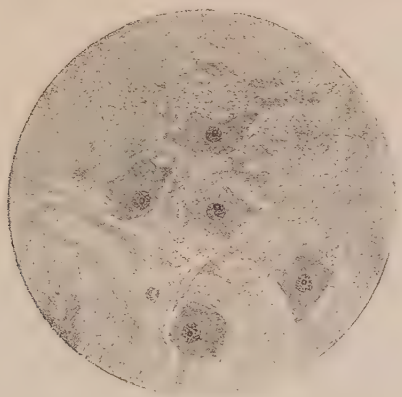

Fig. 204.—Showing Atrophied Cells.

creased). Codeia and morphine give the best permanent results. Quinine and mineral acids are of much service in relieving the vasomotor and sensory symptoms. I have used bromide of uranium (gr. $\frac{1}{80}$) with some apparently good results. Arsenic, Indian hemp, tinct. veratrum viride, salicin, and salicylate of soda rank next in value. Nitrate of silver, conium, curare, bromides, atropia, phosphorus, cod-liver oil, iron, and picrotoxin have all been recommended. They are of doubtful value. Suspension is of some use in a minority of cases not too much advanced. The mind in paralysis agitans is sometimes in an emotional, almost hysterical, condition, and patients are easily made better for a time by some psychical influence. Hypnotism by means of fascination is said to be of use, but it has failed in my experience.

CHAPTER XXVII.

TROPHIC AND VASOMOTOR DISORDERS.

PROGRESSIVE FACIAL HEMIATROPHY is a disease characterized by a progressive wasting of one side of the face.

Etiology.—It begins oftenest in the young between the ages of ten and twenty. Females are more affected.* There is in rare cases a hereditary history. Injury and infectious fevers sometimes start up the trouble. The left side is oftener attacked.

FIG. 205. FACIAL HEMIATROPHY, EARLY STAGE, showing alopecia and osseous depressions.

Symptoms.—The disease begins very gradually and shows itself first in patches. The skin gets thinner, there is loss of pigment, hairs fall out, and the areas may have a yellowish appearance. Sometimes the periosteum and bone are affected, and shallow depressions are formed which may be anæsthetic (Fig. 205). The subcutaneous tissue is most involved, the muscles least, and there are no changes in electrical reaction. The muscles of mastica-

*About 100 cases have been reported. Among 5 seen by myself, 3 were in females, 2 in males. The disease in all cases, so far as could be found, began between the tenth and twentieth years.

tion are usually spared. The bone undergoes general atrophy and the lower jaw may be reduced to two-thirds the normal size. The secretion of sebum ceases, but that of sweat may be increased. The temperature falls. There is a sinking in of the eye, narrowing of the lid, and dilatation of the pupil. There is sometimes pain and rarely anæsthesia. The tongue and other parts of the body may be involved. Spasmodic movements of the muscles of mastication have been noted (B. Sachs). Scleroderma sometimes appears on the face or hands.

The disease progresses rather rapidly at first, but finally comes to a standstill. It does not shorten life.

Pathology.—There has been found a degenerative neuritis involving the fibres of the trigeminus; its descending root and the substantia nigra were atrophied (Mendel).

The diagnosis is easy. Hemiplegia with atrophy in children, congenital asymmetry, and atrophy from gross lesions of the nerve are distinguished by their stationary character or the presence of severe pain.

Treatment.—There is no treatment known to be of service. Tonics, iodide, and electricity may be tried. Dercum has suggested resecting the trigeminal nerve.

Acromegaly (Marie's Disease).

Acromegaly is a chronic dystrophy characterized by gradual enlargement of the hands, feet, head, and thorax, and by a dorso-cervical kyphosis. Though the disease was first described only six years ago by P. Marie, the number of cases reported is rapidly increasing, and if one includes various abortive types it is not extremely rare. Eleven American cases have been recorded.

Etiology.—It affects the two sexes nearly alike. It begins between the ages of eighteen and twenty-six; recently a congenital case has been reported. No hereditary influence or definite exciting cause is known. The patients are sometimes naturally endowed with large extremities.

Symptoms.—The disease begins with a gradual enlargement of the hands, feet, and head. In women there is amenorrhœa, in men sexual weakness; slight rheumatic pains, headaches, malaise, mental hebetude, anæmia, and general weakness are present. The skin is dry and there is polyuria.

The hypertrophy affects the soft parts as well as bones. In these latter there is periosteal thickening and hyperplasia, with the result of producing increase in width more than length. The arms are not much involved, nor is the shoulder girdle, except the clavicle. The lower jaw is much more involved than the cranium. The tongue, lips, and nose are enormously hypertrophied. The thorax is enlarged antero-posteriorly and flattened. There is sometimes dulness over the sternum due to persistence of the thymus. The pelvis may be enlarged, but the hip and leg bones are generally spared. The feet undergo enormous hypertrophy. The following are some of the measurements in the case

that has come under my observation, reported by Adler, and in cases reported by Osborne and Packard:

Length of hand, ...	7.6 to 8¾ inches.
Length of foot, ...	11.7 to 12¼ "
Cranial circumference, . . .	24 to 26½ "
Circumference of thorax, . .	44 "

The vision is sometimes impaired and there may be hemianopsia. The muscles may be at first hypertrophied, later atrophied. There are no paralyses and rarely any anæsthesiæ.

The disease runs a very chronic course, lasting ten or twenty years.

FIG. 206.—ACROMEGALY.

Pathology.—There has been found an enlargement of the pituitary body, and it has been suggested that the disease is due to disorder of its function. But this enlargement is not constant, and tumor of this organ does not cause acromegaly. The attempts to place the disease in relation with a persistent thymus, sclerotic changes of the sympathetic, and disease of the thyroid, all of which conditions have been found, are equally unsuccessful. The disease must be regarded as a perversion of nutrition of unknown origin. The enlargement of the bones is a true hypertrophy, not an inflammation.

The *diagnosis* must be made from congenital enlargements, from so-called giant growth which affects single members, and from osteitis deformans. In the latter disease it is the shafts of the long bones and the cranium, not the face, which are involved.

Pneumogenic osteo-arthropathy is the name given by Marie to a disease associated with pulmonary and pleuritic disease, and

characterized by enlargement of the extremities and peculiar deformities of the terminal phalanges. The enlargements are not uniform. The tongue is not affected. The wrist and ankle bones are hypertrophied, the finger-tips are bulbous and spade-shaped.

Prognosis.—Acromegaly is incurable, but it has been arrested, or at least has ceased to progress, and it may not greatly shorten life.

Treatment.—Cases have been reported in which iodide of potassium and arsenic have arrested the disease. In general, the treatment is only symptomatic.

Angioneurotic Œdema (Circumscribed Œdema).

Angioneurotic œdema is a functional disorder characterized by the rather rapid appearance of circumscribed swellings upon different parts of the body, these swellings being due to disturbances of vasomotor innervation and not of an inflammatory character. The disease occurs oftenest in early adult life, the average age being from twenty to thirty, but it has been observed in young children and even in the aged. It occurs oftener in males than in females, except in this country, where the reverse ratio exists. Hereditary influence plays a part in some cases; the disease has been known to run in families. It occurs oftener in winter and oftener in the early morning hours. Exhausting occupations predispose to it. The exciting causes are sudden exposure to cold, slight traumatisms, fright, anxiety, grief, and the ingestion of certain kinds of food such as apples or fish. A peculiar form of this œdema seems to develop in connection with menstruation.

Symptoms.—The disease appears without much if any warning. In a few minutes or hours there develops a circumscribed swelling upon the face or arms or hands. This swelling varies in diameter from one-half inch to two or three inches. It may be dark reddish or rosy or it may be pale and waxy. It does not easily pit on pressure. There is sometimes a local rise, sometimes a fall in temperature. It is accompanied with sensations of tension and stiffness, scalding, burning, and sometimes itching, but there is no actual pain. The swelling is usually single, but it may be multiple. It is located most often upon the face, next upon the extremities, particularly the hands; next on the body, then in the larynx and throat, and then on the genitals. The swellings last from a few hours to two or three days. Between the attacks the patient feels well. They are apt to return at intervals of three or four weeks to several months. Sometimes they are only brought out by certain peculiar exciting causes, such as indigestion or mental anxieties or emotional disturbances. When the disease attacks the larynx or throat, serious symptoms of dyspnœa and suffocation may appear; surgical interference

may even be called for, and death has been known to result. It has been thought that neurotic œdema may sometimes attack the stomach, producing symptoms of nausea, vomiting, and great gastro-intestinal distress, and an acute neurotic œdema possibly sometimes attacks the lungs. This, however, is unlikely, since the vasomotor innervation of the pulmonary blood-vessels is a very stable one.

The *pathology* of the disease is not known, except that it is unquestionably a disorder due primarily to disturbance in nerve innervation. The nerves affected are, furthermore, undoubtedly vasomotor nerves. The œdema is precisely similar to that which is associated with attacks of tic douloureux and migraine.

Diagnosis.—The symptoms of the disease are so peculiar that they are easily recognized. The spontaneous appearance of the œdema, its recurrence at certain intervals, and the absence of pain and evidences of inflammation are sufficient usually to enable us to recognize it. The giant urticaria is a disease resembling neurotic œdema and probably closely allied to it. The blue and the white œdema of hysterics differs in being persistent and associated with paralyses, anæsthesias, and contractures.

The *prognosis*, so far as cure is concerned, is not very good. The attacks, however, can be ameliorated, and the disease itself is not serious as regards life and the enjoyment of a fair degree of general health.

The *treatment* consists in the adoption of such measures as will give tone and stability to the nervous system; the use of cold baths, exercise, and massage is indicated. Internally mineral acids and strychnine may be of some value. Cascara, nux vomica, the salicylates, arsenic, quinine, and atropine are all drugs which have been recommended.

CHAPTER XXVIII.

THE DISORDERS OF SLEEP.

Insomnia, Hypnotism, Morbid Somnolence, Catalepsy, Trance, Lethargy, the Sleeping Sickness.

Sleep is a condition in which consciousness is normally lost and in which the whole body, but particularly the brain, enjoys functional rest, while constructive and nutritive activity goes on.

Physiology.—The most conspicuous phenomenon of sleep is the subsidence of the higher cerebral functions; yet other organs, notably the muscular system, also take part in the resting process. The brain during sleep is slightly anæmic, the deficiency in blood being a part of, but not the cause of, the phenomenon. The remote cause of sleep is inherent in the nervous tissue itself, which follows the great rhythmical law common to all living tissue of rise and fall in its irritability. It is probable that the immediate cause of drowsiness is the exhaustion of the irritability of the cortical cells and the benumbing of them by the circulation of waste products in the blood. Many facts in the history of the pathology of the brain point to the existence of a sleep centre, which, being especially acted upon, tends to inhibit the consciousness and draw the mind into a somnolent state.

As sleep is only a function, we cannot speak of its diseases, but only of its disorders, and these really form but a part of the diseases of the brain or of general diseases. It is a matter of convenience, however, to discuss some of these separately.

Classification.—Custom has established the use of certain terms for the various disorders of sleep, and such terms must be for the most part adhered to. It will be proper, however, for the sake of completeness, to arrange the various disturbances we are to discuss in accordance with the modern methods of studying the pathological changes of bodily functions. We propose, therefore, the following classification, which indicates the various depressions, exaltations, and perversions of the function of sleep:

I. State of normal sleep. *Hypnosis.*	Somnus.
II. States of absence of sleep. *Ahypnosis.*	Insomnia.
III. States of perverted or artificial sleep. *Parahypnosis.*	Dreams, nightmare, night-terrors, sleep-drunkenness, somnambulism, hypnotism.
IV. States of excessive or frequent drowsiness and sleep. *Hyperhypnosis.*	Morbid somnolence, paroxysmal sleep, epileptic sleeping attacks, trance-sleep, lethargy, sleeping-sickness of Africa.

I. Normal sleep varies much in accordance with age, sex, the individual, and, to a slight extent, with occupation, race, and climate. The infant sleeps fourteen or sixteen hours out of the twenty-four, the adult needs about eight hours, while the aged live healthfully with but six. Women need half an hour or an hour more than men. A few persons, generally men, need nine, ten, or even twelve hours of sleep daily; others require only six. Brain-workers, as a class, take less sleep than laborers. Sleep is sounder and longer in cold climates and among northern races.

II. Insomnia is a term given to conditions in which persons simply suffer from insufficient and restless sleep or from entire absence of sleep for a long time. Such conditions result from a great variety of causes. It is my purpose to discuss only those forms in which the trouble is functional or nutritional, leaving out of consideration the symptomatic insomnia of organic brain disease and that occurring as the result of painful diseases.

An entire absence of the capacity to sleep occurs most often and typically at the onset or in the course of insanity. It is here a prominent and most distressing symptom. The length of time during which a person can live without any sleep is about the same as that during which he can go without food, viz., three weeks. Many hysterical, neurasthenic, or incipiently insane individuals will assert that they have not slept for weeks, but careful examination shows that they have at least been in a drowsy, somnolent condition, which is, in a measure, physiologically equivalent to sleep.

Etiology.—The cases in which persons can only get a troubled rest of a few hours are much more numerous. It is a disorder of the third, fourth, and fifth decades of life. Women are less liable to suffer from it than men, and the laboring classes less than those engaged in business or professional pursuits. A frequent symptom of neurasthenia is an imperfect, and especially an unresting, sleep. In these neurasthenic cases there is a loss of vasomotor tone. In gout and in the so-called latent gout, or lithæmia, insomnia is a frequent symptom. One of the few nervous symptoms of secondary syphilis is insomnia. Insomnia may develop as a bad nervous habit in persons who are neglectful of themselves. It occurs sometimes as an

hereditary neurosis. I am acquainted with a family in which, for four generations, one or more of the members have suffered from chronic insomnia throughout life. In anæmia and chlorosis there is often insomnia at night, combined with somnolence during the day-time. Disease of the heart and arteries may lead to insomnia, and under this head come the cases which occur in Bright's disease with tense arteries and anæmic brains. Disorders of the stomach lead to disturbed sleep oftener than complete insomnia, and the liver, when inactive, causes somnolence rather than the contrary. The poison of malaria and the toxic agents of fever must be added to the list of causes of imperfect sleep.

It will be seen that the causes of chronic functional insomnia may be classed under the following heads:

1. Neurasthenic and vasomotor, including hereditary and habit insomnia.

2. Vascular and cardiac, including heart disease, arterial fibrosis, and general anæmia.

3. Auto-toxic or diathetic, including lithæmia, gout, and uræmia.

4. Toxic, including syphilis, lead, malaria, tobacco, and various drugs, such as coffee, tea, and coca.

In many cases there exists a combination of these causes.

Symptoms.—The forms and degrees of insomnia vary greatly. In children it is accompanied usually with much mental and physical disturbance. The patient is restless, excited, talkative, or querulous and irritable. The insomnic child is more ill than the insomnic adult. In neurasthenic insomnia there is a tumult of thoughts which prevent sleep, or sleep is superficial, unresting, and interrupted by dreams. In many cases of insanity insomnia is characterized by great motor restlessness. In old people insomnia is generally of the quiet kind.

Treatment.—As insomnia in all its phases is often a symptom of some general disorder, treatment of a curative kind must be directed to this. Anæmia, lithæmia, uræmia, malaria, and the other toxic influences must be removed by remedies adapted to these conditions. But besides constitutional treatment there is a symptomatic treatment which will be discussed here.

The older physicians, in treating insomnia, used to depend largely on hyoscyamus, camphor, opium, and the fetid drugs, such as asafœtida, musk, and valerian. Hyoscyamus is still used. It is to be given in large doses, such as five or ten grains of the extract, or even more, and from ten to twenty drops of the fluid extract. The hydrobromate of hyoscin, in doses of gr. $\frac{1}{150}$ to gr. $\frac{1}{100}$ or more, is one of the best forms. Hyoscin is indicated in the insomnia of the insane, especially in forms accompanied with motor activity. Chloral hydrate still holds its own as one of the

surest of hypnotics. The dangers involved in its use have been somewhat exaggerated, though they are sufficiently real. Doses of gr. x. and gr. xv. are often quite large enough, but in alcoholic insomnia it may be given in twice the above amounts, guarded with ammonia and digitalis. Not a few persons find that chloral has bad effects. The patient awakes with a dull, heavy sensation in the head, slight headache, or gastric disturbance.

The various bromides are efficient and safe hypnotics if properly used. The immediate effect of them is simply sedative, and sleep is not produced unless very large doses are given. Some persons are even kept awake by average doses (gr. xv. to xx.). In insomnia, therefore, bromides are best prescribed in doses of gr. xv. three times a day. By the second evening sleep is generally secured. The bromide habit is rarely formed, and is in itself not so seriously injurious as the chloral or opium habit. The bromides alone are hardly strong enough hypnotics for alcoholic insomnia or the insomnia of insanity. I have found them to fail in the insomnia of the aged. Paraldehyde ranks close to chloral in its value as a hypnotic. In some persons it disturbs the stomach, but not in all, and it may be used as a hypnotic for months without its power being impaired. It is a disagreeable drug, and there is nothing, so far as I know, that palliates its offensiveness. I prefer, however, to prescribe it in ʒ i. doses poured upon a teaspoonful of powdered sugar. Doses of ʒ ss. are sufficient to cause sleep in many cases, and collapse may be caused by ʒ ij. given to weak patients. Urethran, in doses of a scruple or more, is a mild and agreeable hypnotic, but not so certain as paraldehyde. Amylene hydrate is a hypnotic of similar properties to paraldehyde, but less disagreeable. It is given in doses of about one drachm. Lupulin in large doses, gr. x. to gr. xx., is a good hypnotic. Sulphonal in doses of gr. x. to gr. xxx. given two to four hours before retiring is one of the best hypnotics. Chloralamide is more agreeable and safer than chloral, though it acts practically in the same way. The dose is ℈ i. to ℈ ij. Somnal, trional, tetronal are new drugs which act well in many cases.

Among the antispasmodics are several drugs which occasionally answer well in the insomnia due to nervous irritability. A drachm of the fluid extract of valerian or of spirits of lavender, for example, may be prescribed. In some forms of insomnia—perhaps best in those due to fever, or pain, or some rheumatic or gouty trouble—antipyrin in twenty-grain doses acts well. It is known that in many cases of mild types of insomnia a dose of whiskey, brandy, or beer will put the patient to sleep. Besides drugs, there are many hygienic or mechanical measures to which the physician may successfully resort—listening to monotonous

noises, reading dull or heavy books, counting, or keeping before the fancy some blank or wearying picture—

> "A flock of sheep that leisurely pass by
> One after one; the sound of rain and bees
> Murmuring; the fall of rivers, winds and seas,
> Smooth fields, white sheets of water, and pure skies."

Mechanical remedies have nearly all for their purpose the withdrawal of the blood from the brain to the skin and abdominal viscera. Hot foot-baths or warm general baths, cold douches down the spine, beating the limbs with rubber hammers, brisk exercise, a light meal, massage, all are at times efficient hypnotics. Persons who suffer from insomnia should sleep in cold rooms, the head should not be too high nor very low, and in most cases they are better without late suppers, even though these be light. Mental work should be laid aside several hours before retiring, and the evening devoted to quiet conversation and reading or amusements that do not actively excite the nerves. Many persons live in good health though they sleep in the day and stay awake at night. Journalists and editors, whose work obliges them to go to bed in the early morning, often continue for years without impairment of physical vigor. Yet despite this, it is true that the best time for sleep is at night, and that the old maxim, "early to bed," is a sound one. The human system requires a certain amount of sleep and should have it. The industrious and ambitious often try to train themselves to shorter hours, but though they may succeed for a time, nature will not be cheated out of her due and health suffers in the end. It is a wide-spread custom in some countries to take a short nap in the day-time, and the custom is a good one. America has not adopted it, but might do so with benefit to the health of her brain-working class. Many from childhood up do not get a sufficient amount of sleep.

III. Perversions and Disturbances of Sleep.—Sleep is said to reach its deepest stage in from one to two hours after it begins. There is then after this a gradual lessening of the depth of sleep. Probably there are great variations in this rule, for many persons seem in soundest slumber several hours after falling asleep. But, at any rate, there are slighter stages of sleep at its inception and toward its end. These are the favorite times for dreams, and at this period also there develop the peculiar phenomena of sleep-drunkenness.

Dreams, Nightmare.—When sleep is perfect and profound, dreams afterward remembered do not occur. Dreaming is, therefore, a morbid symptom, although often of trivial significance, especially if it occurs at about the time of natural waking, when slumber is, in its physiological course, passing into the lighter stages. In sleep, no matter how light, the action of the regulat-

ing centre which directs thought, controls emotion, and exhibits itself in volition is suspended; the psychical mechanism, if excited to action at all, works without purpose, like a rudderless ship at sea. Ideas and emotions succeed each other by the laws of association, but are not properly correlated, and judgment and logical reasoning are gone. As a rule, dreams are made up of somewhat ordinary ideas and fancies incoherently associated, and shifting too rapidly to call up much feeling. When from some point in the body painful sensory excitations do excite disagreeable images, emotions of a most violent kind may be felt.

In the earlier stages of civilization, among primitive people, dreams were comparatively rare. When they did come with vividness they were regarded with importance, and often were considered visitations of spirits. Civilized man dreams more, but he has learned to treat his fancies with corresponding indifference. The attempts of scientific men to formulate laws regarding them have been productive of small results. Some diseases, however, produce, as a rule, dreams of a more or less peculiar kind. Thus heart disease is accompanied with dreams of impending death. Previous to attacks of cerebral hemorrhage, patients have dreamed of experiencing some frightful calamity or of being cut in two. Intermittent fever is often announced by persistent dreams of a terrifying character. Hammond has collected a large number of what he terms prodromic dreams, all going to show that before recognizable signs of disease are present morbid dreams of various kinds may occur. Albers says: "Frightful dreams are signs of cerebral congestion. Dreams about fire are, in women, a sign of impending hemorrhage. Dreams about blood and red objects are signs of inflammatory conditions. Dreams of distorted forms are frequently a sign of abdominal obstructions and diseases of the liver."

Nightmare is a disorder incident to the hypohypnotic state, or that of incomplete sleep. It is one of those minor ills that are nearly always symptomatic of an irritation in some part of the body. The usual causes of it are some digestive disturbance (repletion) and cardiac disease. Persons of a nervous temperament are more subject to it; and there are individuals whom it makes suffer all their lives. The popular belief that sleeping on the back favors it is, in general, a correct one. When nightmare occurs in cardiac disease a certain position, semi-recumbent or on the right side, must be maintained, or the painful fancies awaken the patient. Healthy people can get sound sleep whether lying upon the back, the side, or the stomach. But light sleepers, and those with sensitive abdominal viscera, generally find that the position on the right side is the most comfortable and less provoca-

tive of unpleasant dreams. Prolonged mental or physical strain, excitement, and worry predispose to nightmare. Farinaceous foods, excessive use of strong liquors, coffee, and tobacco, all have a similar tendency. Nightmare occurs also in anæmia and malaria, and it may, in fine, be excited by morbid conditions in any part of the body. It sometimes occurs about the menstrual period in women. Its most common feature is a sense of suffocation or impending death.

Pavor nocturnus, or night-terrors, is a sleep disorder peculiar to children. It is allied to nightmare on the one hand and sleep-drunkenness on the other. It differs from the former condition in that the child continues to suffer from the distressing fancies for some time after he is awake. Night-terrors occur usually one or two hours after sleep has begun. The child wakes up screaming with fright, and perhaps runs about the room or seeks its parents for protection against some imagined harm. The disorder occurs in weakly, anæmic, nervous, or rheumatic children. It is due sometimes to lithæmia, or, as the older writers put it, rheumatism or gout of the brain. Digestive disturbances, worms, dentition, hereditary syphilis, mental strain, fright, and excitement are placed among the causes. It sometimes appears to be a paroxysmal neurosis allied to epilepsy. The disorder is usually harmless and the prognosis favorable.

Somnolentia, or sleep-drunkenness (*Schlafentrunk*), is a condition of incomplete sleep in which a part of the faculties are abnormally excited while the others are buried in repose. It is a kind of acted nightmare. The person affected is incoherent, excited, and often violent. He experiences the delusion of some impending danger, and while under it acts of violence have been committed. The condition is one of medico-legal importance, therefore, and has been discussed by writers on that science (Wharton and Stillé). Minor degrees of it are often noticed in children and in adults who are roused from a very profound sleep. It at times becomes a habit, and a most annoying or dangerous one. The disorder in its severe form is fortunately very rare.

The treatment of morbid dreams, nightmare, and pavor nocturnus must be directed to a removal of the causes. Tonics, cardiac stimulants, laxatives, anti-rheumatics, attention to diet, are called for according to the condition of the patient. Change in surroundings is often necessary. Among symptomatic remedies the bromides are the best, except in lithæmia, when alkalies and salicylates may prove more serviceable. In somnolentia the patient should be prevented from getting into too profound sleep. He may be awakened once or twice during the night, or take a nap in the day-time. The head in sleeping should be raised high and the body not too heavily covered.

Somnambulism.—Somnambulism is a condition similar to hypnotism or the mesmeric state. In it volition is abolished, and the mind acts automatically under the dominance of some single idea. Sight, hearing, and nearly all the avenues of sense are closed. The sleep-walker avoids obstacles and performs ordinary acts automatically, like an absent-minded man, which in reality he is. All those mechanisms which have been trained by constant repetition to act automatically, like that which preserves equilibrium, are active, and their powers may even be heightened, so that the somnambulist may walk along roofs or on dangerous roads and thread intricate passages without harm. The automatism of the somnambulist may continue for hours, until a journey has been performed or a task completed. He may carry out with success intricate mathematical calculations, write a letter, or work upon a picture, but he only follows along the lines established by constant iteration in his waking moments. He can originate nothing new. He is roused from his state with difficulty, and when out of it he remembers nothing of what has occurred.

Somnambulism usually arises from overeating. Sleeping with the head too low is another cause. Violent emotions act indirectly by disturbing digestion. The habit being once established, however, attacks occur without apparent cause. The disorder occurs oftenest in young people about the age of puberty, and it then attacks the sexes alike. Later in life women are more often affected. The disease is fostered sometimes at school by the attentions of the schoolmates. In most cases a condition of morbid sensitiveness underlies it. The patients are neurotic. Hereditary somnambulism has been observed. Its attacks have alternated with those of catalepsy. They are likely, after a time, to become periodical, occurring every week, fortnight, or month. The somnambulic state may come upon a person in the daytime. It is then regarded as spontaneous trance, or hypnotism. It is not the case, however, that persons who are easily hypnotized are usually somnambulists, though the reverse may be true.

Somnambulism is a term that should include not only sleep-walking, but sleep-talking.

The treatment of somnambulism is very much like that for sleep-drunkenness. The patient's surroundings must be investigated, and unfavorable influences, such as may occur at school or from injudicious nurses, be removed. He should be prevented from sleeping too soundly, the head should be raised, the clothing light, the diet regulated. Remedies like iron, quinine, phosphorus, and cod-liver oil may be given. When the patient is discovered in the somnambulistic state he should not be awakened, or at least not until he is safely back in bed.

HYPNOTISM, TRANCE, MESMERISM.—*Hypnotism* is a morbid mental state artificially produced and characterized by (1) perversion or suspension of consciousness; (2) abeyance of volition; (3) a condition of suggestibility leading the patient to yield readily to commands or external sense impressions; and (4) intense concentration of the mental faculties upon some idea or feeling.

The proportion of persons of all ages found to be hypnotizable by Beannis was about eighteen or twenty per hundred. Children up to the age of fourteen are very susceptible. After the age of fifty-five susceptibility lessens. Men are almost as easily affected as women; but persons of a docile mind and those trained to some degree of mental discipline and capacity for submission, such as soldiers and artisans, are more sensitive. In this country the percentage of hypnotizable subjects is less than it is in Europe. Hysterical and insane persons are not very susceptible. Those who have been mesmerized once are more easily affected afterward, and may even pass into the state involuntarily.

Methods.—There are two ways of inducing hypnotism, the fixation method and the suggestive method. The former and older plan, devised by Braid, is to make the patient fix his eyes for five to ten minutes on some bright object at a distance of six to eight inches from the eyes and a little above the horizontal plane of vision. A modification of this is the fascination method of Luys, by which the patient is made to fix his eyes at revolving mirrors.

In the "suggestive method" devised by Liebault and Bernheim the subject is placed in a chair in front of the operator. The operator then talks to the subject in a firm and confident voice, assuring him that he will go to sleep in a short time, telling him to make no resistance, that his sleeping will be natural, that nothing will be done to worry or fatigue him, that he will dream pleasant dreams, that he will wake up feeling better; then that he is feeling drowsy, his eyes are heavy, objects look confused, the lids are falling, they are closed—in a moment more the patient goes off to sleep. This requires some little time—five to fifteen minutes. It may fail the first time and succeed the second.

Hypnotic states may be self-induced by rigorously fixing the attention upon some object. The ecstatic states of the saints and the nirvana of the Buddhists are forms of hypnotism; so also are the trance states into which some clairvoyants and spiritualistic preachers place themselves; this same curious phenomenon is at the bottom of the so-called "mind-healing" science, and it enters into rational therapeutics and orthodox religion. The capacity of the human mind for hypnotism or semi-hypnotic states is, therefore, a most curious and important fact.

Symptoms.—The person who has been hypnotized at first sits or lies quietly in the position he had assumed during the manipulations of the operator. No notable physiological changes occur, as, for example, in the pulse, respiration, temperature, pupils, skin, etc. Some increase in the cerebral blood supply, however, is said to be present. The patient will now respond automatically to any outside command or will be dominated by any idea which is suggested to him. He will talk, or walk, or run, or gesticulate, assume expressions of fright, anger, or joy, entirely in accordance with the command given. Apart from these commands he is entirely dead to the outside world. He hears, sees, smells, tastes, and feels nothing. He can be burned, cut, or injured without showing any signs of feeling. At a suggestion he may be made cataleptic, somnambulic, or paralytic. This state is termed *somnambulistic trance.* If left to himself, he gradually sinks into a deep sleep, from which he can with difficulty be roused. After a time, rarely more than one or two hours, he awakes as from ordinary slumber. This latter state is called trance-coma, or *lethargic hypnotism.* The attempts of the Charcot school to divide hypnotic phenomena into three forms, the somnambulic, cataleptic, and lethargic, are hardly successful. Sensitive subjects can be thrown at once into lethargy, catalepsy, or somnambulic states at the command of the operator.

The phenomena of hypnotism depend upon the wonderful sensitiveness and quickness of the subject in responding involuntarily, with all his nervous energy, to outside suggestion. Dishonest persons may learn the latter trick and thus simulate the hypnotic state. Travelling mesmerizers utilize such persons largely, hence no confidence can be placed in the phenomena exhibited by them.

Besides the above hypnotic state, which I call the major hypnotism, various minor stages occur. These are produced by the "suggestive method" of hypnotizing. By this latter plan patients are thrown into various degrees of the hypnotic state from slight drowsiness to complete lethargy.

Patients naturally come out of the mesmeric state through the channel of deep sleep or lethargy. Ordinarily they are dehypnotized by word of command, or by a pass of the hand, or any impression which the patient expects to be used for the purpose.

Hypnotized persons have been observed to have a diminution in the spinal reflexes and a muscular hyperexcitability. They sometimes show a most extraordinary exaltation of visual, auditory, or other special sense.

Pathology.—The underlying changes of the hypnotic condition are unknown and will probably long remain so. Hypnotism is no doubt associated with changes in the vascularity of

different parts of the brain and with rapid breaking-down of nerve tissue. Animals constantly subjected to hypnotic influence become demented (Harting, Milne-Edwards).

The state of major hypnotism is probably pathological. It is a neurosis. Minor hypnotic states are but slightly removed from the normal, and their production is not injurious.

Diagnosis.—As hypnotic states may be imitated and as injuries or crimes may be done during this state, it is very important to be able accurately to distinguish it. Since the phenomena are all subjective, this is very difficult. The methods of value are these: 1. Careful examination of the general phenomena by experts while the subject is in the alleged hypnotic state. 2. Testing the muscular hyperexcitability by percussing motor points. 3. Tests of alleged anæsthesia by sudden burning, or pinching, or injuring the subject. 4. Tests of the tetanic muscular rigidity by the revolving tambour. In the hypnotic state the hand may be extended and held with perfect steadiness, while in conscious states a tremor soon appears. 5. Tests with glasses and other apparatus may be made to determine alleged anæsthesiæ of the special senses.

Therapeutics.—The practice of using major hypnotization is injurious, tending to exhaust the nervous force and weaken the will. It should be done only with the greatest care. Its utility in therapeutics I greatly doubt. It may relieve symptoms in the hysterical for a time, but it cannot be of permanent benefit and is likely to lead to actual harm.

The induction of minor hypnotic states by suggestion is not harmful if carefully and moderately employed. Its practical results, however, are not great, and the method is tedious, uncertain, and sometimes ridiculous. It has its value in pedagogy, among children, in neurasthenia and morbid habits. The general popularization of hypnotism by means of mind-cures, Christian science, etc., accomplishes its results at the expense of mental demoralization; and faith-healing institutes are more pernicious elements in society than gin-mills.

Morbid Drowsiness.—This is a very common symptom, which may be due to any one of the following causes: 1. Old age, when there is a weakened heart or diseased arteries, with cerebral mal-nutrition. 2. The diseased vascular conditions which precede cerebral hemorrhage. 3. The cerebral mal-nutrition or inflammations occurring before or during certain forms of insanity. 4. Various toxæmiæ, *e.g.*, malarial, uræmic, cholæmic, and syphilitic. 5. Dyspepsia and gastric repletion. 6. Diabetes. 7. Obesity. 8. Insolation. 9. Cerebral anæmia and hyperæmia. 10. Exhausting diseases. 11. Concussion of the brain. 12. Climatic conditions, cold, etc.

A very common cause of drowsiness is dyspepsia attended with some torpidity of the liver, the condition popularly known as "biliousness." Another frequent cause is malarial infection, which perhaps acts indirectly by impairing the functional activity of the liver. Drowsiness from these causes oftenest comes on in the afternoon. Anæmia is attended with drowsiness during the day, while there is often insomnia at night. Syphilis is more likely to cause insomnia, but in its third stage somnolent conditions may be produced which are of serious significance. Drowsiness occurs from the effects of severe cold. It sometimes develops when persons change their surroundings, especially on going to the seashore, for low levels and a high degree of atmospheric pressure seem to promote sleep. The drowsy state that sometimes follows concussion of the brain is a familiar phenomenon. Some persons, no doubt, acquire the habit of drowsiness. At first the trouble may have been induced by indigestion, "biliousness," or malarial infection, but it persists after the cause is removed. Such persons can hardly sit through a lecture, a church service, or any exercise requiring quiet and attention. As the morbid drowsiness here described is only symptomatic, its treatment need not be discussed. Such remedies as coca, coffee, tea, atropia, glonoin, do not produce results equal to expectations.

Morbidly Deep Sleep.—Certain persons, when they sleep, pass into an almost lethargic slumber. Persons who sleep in this way often sleep a longer time than normal. They are awakened with difficulty, and then suffer with headache or disagreeable sensations throughout the day. The symptom may be a prodroma of insanity. Instances in which persons retire at the usual hour, but can with great difficulty be roused in time for the ordinary duties of the day, are not rare. Some of these are illustrations of the vice of indolence, but in other cases there is an absolute need of nine, ten, or even fourteen hours of sleep.

This disorder of sleep is most liable to occur in the young and in those of nervous temperament. It often seems to be a congenital condition, for which nothing can be done. In other cases it results from over-feeding and indolent habits. Treatment is much the same as that indicated for sleep-drunkenness and somnambulism.

3. *Paroxysmal Sleep, Narcolepsy, Sleep-Epilepsy.*—It sometimes happens that persons suffer from sudden attacks of unconquerable drowsiness; they fall off into slumber despite every effort of the will. These are more than drowsy sensations, for sleep, or a state resembling it, cannot be kept off. Some of these cases are of a purely nervous character, *i.e.*, the trouble is not due to a humoral poison or to organic disease, but to a paroxysmal change in the nervous centres of a vascular or chemical

character, causing sleep. It may be that the patient is epileptic and the sleep-seizure takes the place of the ordinary epileptic spasms.

Cases of epileptic sleep, or narcolepsy, and allied forms are not of frequent occurrence. Females are rather more often affected than males, and the susceptible age is from fifteen to forty. The disorder is brought on sometimes by fright, over-strain, and humoral poisons acting on a predisposed nervous system.

The course is chronic and relief is not always obtained. It should be remembered that syphilis, malaria, or anæmia, and indigestion may be elements in the trouble which are important, if not fundamental. Bromides in small doses are often useful

Fig. 207.—A Case of Catalepsy.

factors in treatment. Change of occupation, of mode of life, or of climate may be essential to a cure.

Catalepsy, Trance, Lethargy.—Most of the so-called cases of prolonged sleep, lasting for days or weeks, are cases of spontaneously developed mesmeric sleep in hysterical women or cases of incipient insanity (katatonia or stuporous melancholia). The phenomena in these cases may take the form of *catalepsy*, with waxy rigidity of the limbs, or *lethargy*. In cataleptic states the limbs may be placed in various positions and will remain there for several minutes (Fig. 207). In lethargy or trance states the patient may be plunged into a deep and prolonged unconsciousness, lasting from one day to several years. These are the "sleeping girls" of the newspapers. Others are persons of a too ready susceptibility to mesmeric suggestion, who get into a morbid habit of going into mesmeric sleep spontaneously. In these states there may be a lowering of bodily temperature,

slowing of respiratory and heart action, and excessive sluggishness of the action of the bowels. The patients can hear and may respond to suggestions, but they are apparently insensible to painful impressions and do not appear to smell, taste, hear, or see. The eyes are closed and turned upward, and the pupils contracted as in normal sleep. Many variations, however, occur in the physiological phenomena of these states.

The duration of the attacks of trance lethargy is from a few hours to ten years. Ordinarily, however, profound trance sleep lasts not more than a few days, while those cases in which the sleep is from mesmeric suggestion last but a few hours.

The katatonic cases after a few weeks or months gradually awake, become excited, and then pass into a condition of dementia or into catalepsy again.

5. Morbid Sleep from Organic Disease.—Prolonged and excessive sleep occurs as the result of syphilis of the brain, brain tumors, and the degenerative changes in old age and insanity. Morbid somnolence and stupor are not very frequent in cerebral syphilis, but are quite characteristic. The patient in some cases lies or sits all day in a semi-soporous state; in other cases he walks about, but continually falls asleep at his task. This state of partial sleep may pass off or end in complete stupor (Wood). It does not necessarily signify a serious issue, even though it last for weeks. Somnolence or sleep is a rare symptom in cases of cerebral tumors other than syphilitic. Conditions of drowsiness or stupor have been noted especially in tumors of the corpora quadrigemina and the parietal lobes (Putnam-Jacobi).

Organic diseases of the brain tend to produce conditions of mental weakness, hebetude, or comatose states, rather than anything allied to sleep.

The Sleeping Sickness, Sleeping Dropsy, Maladie du Sommeil.—This is a peculiar disorder, apparently infectious in character, which occurs among the negroes of the western coast of Africa. The disease has been transported to other regions, but is endemic only in Africa. It begins gradually with some headache and malaise. Soon there is felt a drowsiness after meals. This increases until the patient lies for nearly the whole time in a stupor. When awake he is dull and apathetic. There seems to be no fever, and the temperature may even be subnormal; the pulse, too, is not rapid; the skin is dry, the tongue moist but coated, the bowels regular. The eyes become congested and prominent. The cervical glands are enlarged. The disease ends in coma and finally death. Recovery rarely occurs. Sometimes the course of the disease is more violent, and toward the end there are epileptic convulsions and muscular tremors. Autopsies have revealed no definite pathological changes.

Accidents of Sleep.—Owing to the fact that sleep is a resting state of the organism, and that many of its functions are lowered, or their cerebral control lessened, peculiar crises, or physiological and pathological disturbances of nervous equilibrium, occur. Attacks of gout, of asthma, and of pulmonary hemorrhage are most liable to occur during the early morning hours. Deaths and suicides occur oftener in the forenoon, but births oftener at night. Epileptic and eclamptic attacks occur with much frequency at night. Involuntary emissions of spermatic fluid, orgasmic crises, and incontinence of urine are among the pathological incidents of sleep.

Disorders of the Prædormitium.—Sudden attacks of starting of the whole body, shock-like in character, accompanied with peculiar feelings in the head or occiput, not infrequently attack persons as they are dropping off to sleep. They are of slight significance.

CHAPTER XXIX.

CRANIO-CEREBRAL TOPOGRAPHY.

THE object of cranio-cerebral topography is to map out upon the scalp the underlying fissures, convolutions, and other parts of the brain. As this is for purposes of surgical operations, the mapping is done upon the shaved aseptic scalp, with a soft anilin pencil dipped in strong carbolic solution (1 to 4), or with a brush and carbolized tincture of iodine. The only instruments needed are a steel tape measure and an instrument, of nickel-plated soft iron. This consists of a flat strip 25 cm. long and 1 cm. wide. From its middle there branches a second strip 10 cm. long making an angle of 67° with the longer strip.* Practically, the principal points to be determined are the position of the longitudinal, Rolandic, Sylvian, and parieto-occipital fissures and the lower outline of the brain.

The measurements are based chiefly upon the known relations of certain landmarks on the skull to the parts beneath. These landmarks are the glabella, bregma, lambda, stephanion, asterion, and pterion, which are points at the junction of the various sutures with each other and with certain ridges or protuberances. Their position is shown in the cut (Fig. 208) except that of the glabella or prominence just above the naso-frontal suture. The inion is identical with the occipital protuberance.

The following rules are based upon the observations of Heftler, Thane, Reid, Horsley, Fraser, and myself:

I. The longitudinal fissure. This corresponds with the naso-occipital arc.

II. The fissure of Rolando. Measure the distance from the glabella to the inion; find 55.7 per cent of this distance, and the figures obtained will indicate the distance of the upper end of the fissure of Rolando from the glabella. It should be about 48 mm. behind the bregma in male adults, 45 mm. in women, 30 to 42 mm. in infants and young children respectively.

The fissure runs downward and forward for a distance of about 10 cm. measured on the scalp, the real length being about 8.5 cm. The fissure makes an angle of about 67° with the ante-

* Special instruments called cyrtometers have been devised by Wilson and Horsley, but are not necessary.

rior part of the longitudinal fissure. This direction is determined by the instrument above described or by the cyrtometer. The lower third of it is more vertical, and the lower end is 25 to 30 mm. behind the coronal suture. A line from the stephanion to the upper part of the asterion should about pass through it. The fissure is shorter in children.

III. The fissure of Sylvius runs nearly horizontally, and lies either under or a little above the uppermost part of the parieto-squamous suture. *This suture*, the *external orbital process*, and the *parietal eminence* are the guiding landmarks by help of

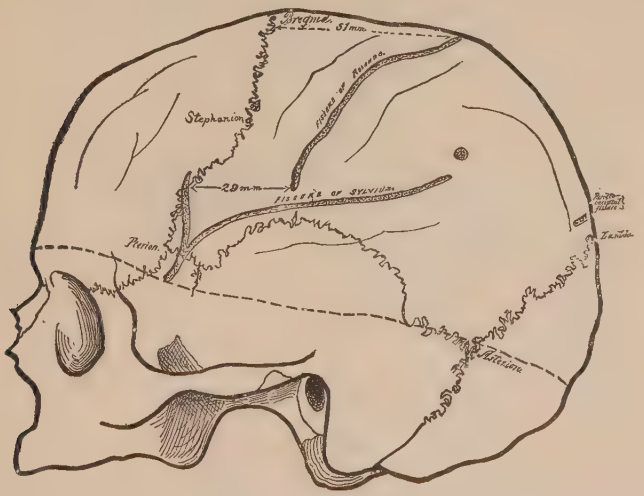

Fig. 208.—Showing the Position of the Bony Points on the Cranium, the Sutures, and the Principal Underlying Fissures, also the Basal Outline of the Brain (after Fouillehouse).

which the surgeon can often operate without marking down lines on the scalp. In children the fissure is sometimes higher and more oblique.

To outline it, draw a vertical line from the stephanion to the middle of the zygoma. Draw a horizontal line from the external angular process to the highest part of the squamous suture; continue this back, gradually curving it up till it reaches the parietal eminence. The junction of the two lines will be at the beginning of the fissure of Sylvius. The vertical line indicates nearly the position of the ascending or vertical branch of the fissure, which is, however, directed a little more forward, and is about 2.5 cm. (1 inch) in length. The posterior part of the line indicates the position of the posterior branch of the fissure.

Reid's method of finding the fissure of Sylvius is to "draw a line from a point $1\frac{1}{4}$ inches behind the external angular process to a point $\frac{3}{4}$ inch below the parietal eminence. The ascending branch starts from a point $\frac{3}{4}$ inch back from the anterior end of this line, and 2 inches (5 cm.) back of the external angular process."

IV. To outline the parieto-occipital fissure, find the lambda, mark a point 3 mm. anterior to it, draw a line through this at right angles to the longitudinal fissure, extending about 2.25 cm. (1 inch) on each side of the median line. This marks the position of the fissure. If the lambda cannot be felt, its position may be found by measuring the naso-occipital arc and taking 22.8 per cent of it. This indicates the distance of the lambda from the inion or external occipital protuberance. The average distance in male adults is 7.42 cm. ($2\frac{7}{8}$ inches). It is greater in women than in men by a little over a millimetre.

V. To outline the frontal lobes: The anterior end of the frontal lobes reaches to a point determined by the thickness of the frontal bone. This ranges from 2 to 8 or more mm. ($\frac{1}{12}$ to $\frac{1}{3}$ inch). The floor of the anterior fossa reaches in front to a level a little above the supraorbital margin (16 mm., $\frac{3}{4}$ inch—Heftler). It slopes down and backward, its posterior limit being indicated by the lower end of the coronal suture.

VI. To outline the temporal lobe and the lower border of the cerebrum: The temporal lobe is limited above by the fissure of Sylvius, below by the contour line of the lower border of the cerebrum. This latter corresponds to a line drawn from a point slightly (about 12 mm.) above the zygoma and the external auditory meatus to the asterion, and continued on along the superior occipital curve to the inion. The anterior border of the lobe corresponds to the posterior border of the orbital process of the malar bone.

The temporal lobe is about 4 cm. ($1\frac{5}{8}$ inches) wide at the external auditory meatus. A trephine, as Bergmann states, placed half an inch above the meatus would enter the lower part of the lobe. The middle of the lobe is in a vertical line from the posterior border of the mastoid process. A line from the upper end of the fissure of Rolando to the point of the process would pass through this important sensory area (Fig. 208; see also p. 351).

VII. To find the position of the central ganglia, viz., corpus striatum and optic thalamus, draw a line from the upper end of the fissure of Rolando to the asterion, practically a vertical line. This limits the optic thalamus posteriorly. A vertical line parallel to the first, a little in front of the beginning of the fissure of Sylvius, limits the corpus striatum anteriorly. A horizontal plane 45 mm. ($1\frac{3}{4}$ inches) below the surface of the scalp at the bregma

limits the ganglia superiorly. The ganglia lie about 35 mm. ($1\frac{3}{8}$ inches) below the superior convex surface of the brain (Féré).

VIII. To reach the lateral ventricles: A number of routes may be taken. The lateral is recommended by Keen. Mark a point $1\frac{1}{4}$ inches behind the external auditory meatus and $1\frac{1}{4}$ inches above a base line made by drawing a line through the lower border of the orbit and the external auditory meatus. Trephine at this point and plunge the director into the brain in

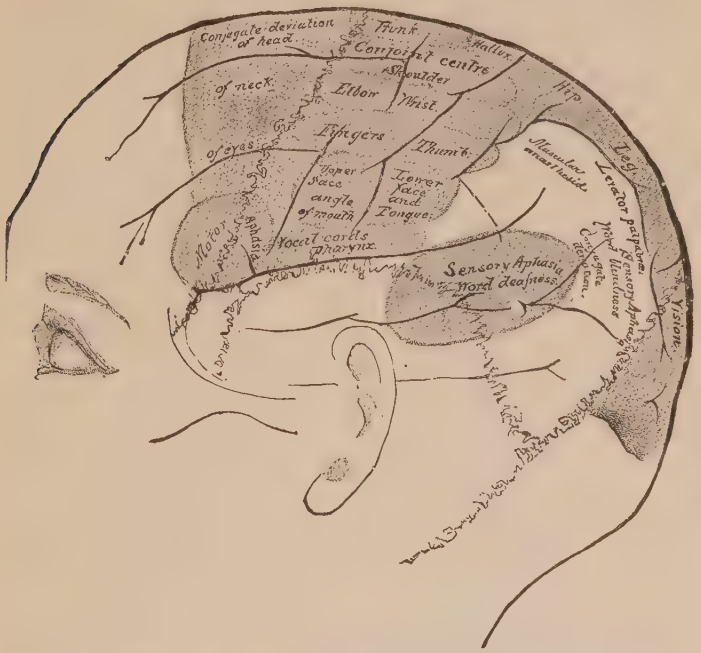

Fig. 209.—Showing the Relative Position of the Fissures and Cortical Centres of the Brain.

the direction of a point $2\frac{1}{2}$ to 3 inches vertically above the opposite external meatus. The ventricle lies at a depth of 2 to $2\frac{1}{4}$ inches (5 to 5.7 cm.).

Mr. Alec Fraser has devised a way of mapping out the fissures by means of a series of composite photographs, so taken as to show the relation of the underlying parts to certain tapes tacked upon the skull. One of his figures is reproduced here (Fig. 210).

In applying this method the surgeon tacks the tapes on the shaved scalp. Then looking at the diagram he finds where the point in the brain is that he wishes to reach and notes its rela-

tion to the median lateral or circumferential tapes. Then as the circumference of the illustrated head is to the circumference of the living one, so is the position of the area on the tapes in the illustrated head to the desired position of the same area in the living one. The illustration is a composite of several adult heads varying in circumference from 20¼ to 23½ inches.

The tapes are divided into inches and half inches. The primary tape is the circumferential passing horizontally round the vault of the head (on the shaven scalp) from the root of the nose (glabella) to the maximum occipital point, which is about one inch above the inion. The tape which thus entirely surrounds the head is divided into four equal parts. The points where the division is made are at the anterior and posterior poles and midway on each lateral half of the tape. From these lateral midpoints a tape is run vertically over the top of the head, and other tapes are run anteriorly and posteriorly half-way between the point where the transverse tape crosses the sagittal suture and the anterior and posterior poles. Another longitudinal tape is run from the anterior to the posterior pole half-way between the sagittal suture and the circumferential tape.

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